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RETINAL TUMORS IN TUBEROUS SCLEROSIS

REVIEW OF THE LITERATURE AND REPORT OF A CASE, WITH SPECIAL
ATTENTION TO MICROSCOPIC STRUCTURE

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Involvement of the retina in cases of tuberous sclerosis escaped notice until 1921, when van der Hoeve,[1] of Amsterdam, Netherlands, published a clinical report describing six cases of tuberous sclerosis in which tumors of the retina were observed through the ophthalmoscope. Since then a number of clinical reports of this condition have appeared, indicating that such retinal tumors are not uncommon and that disturbance of vision and the ophthalmoscopic examination may give the first clue as to the disease.

The case presented here is the sixth in which microscopic study of the retinal tumor is described and the first to be reported in this country in which such a study was made.

REVIEW OF THE LITERATURE

The term tuberous sclerosis, or tuberose sclerosis, was first used by Bourneville, who, in a series of articles published from 1880 to 1898, described a rare form of cerebral sclerosis observed at autopsy in young persons who had shown during life mental deficiency and epilepsy. He noticed in some the coexistence of tumors of the kidney of a primitive, mixed cell histologic character. He also noted the coexistence of adenoma sebaceum, which had previously been described as a hyperplasia of certain elements of the skin distributed in butterfly fashion on the face and often associated with epilepsy and poor mentality.

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1. van der Hoeve, J.: Augengeschwülste bei der tuberösen Hirnsklerose (Bourneville), Arch. f. Ophth. **105**:880-898, 1921.

Largely from the work of Vogt in 1908,[2] the clinical syndrome of mental deficiency, epilepsy and adenoma sebaceum was established, and several series of cases of this condition were published. Adenoma sebaceum may be described as a nodular reddish rash distributed in a butterfly-shaped area over the nose and cheeks. It was soon recognized that the cerebral condition might exist without one or even any of the classic clinical signs. It is also evident that cases of this condition may be brought to clinical notice as instances of tumors of the kidney, heart disease or, because of the disfiguring cutaneous lesions, adenoma sebaceum. This is so because multiple tumors of mixed, undifferentiated cells may be present in the heart, kidney, spleen and other organs, of which it is now known that the eye is one.

Epiloia has been used by psychiatrists to denote the complete syndrome of mental defects, epilepsy and adenoma sebaceum.

The term tuberous sclerosis may be used to include also any combination of aggregations of multiple tumors of this type, although it really describes only that feature which is most constant, multiple tumors of the cortex of the brain.

A family history of developmental defects and psychopathic trends is common, and the disease becomes manifest during the first two decades of life. It is a heredofamilial disease.

Van der Hoeve[1] first described the occurrence of flat tumors in the retina of each eye in six cases, in one of which there was a tumor of the optic disk bulging forward into the vitreous. Others have reported such tumors, but in a much smaller proportion of cases. Critchley and Earl[3] found them in only one of their twenty-nine cases.

They are described as oval or circular white or gray areas in the retina, away from the disk, the average diameter being about one-half that of the disk. Only exceptionally are they in close relation to the blood vessels, and there is no surrounding tissue reaction. There are also in some cases larger nodular tumors, extending into the vitreous, usually at or near the disk. In one of van der Hoeve's[1] cases and in cases reported by M. Nitsch,[4] E. Kuchenmeister,[5] Bau-Prussakakowa[6]

2. Vogt, H.: Zur Pathologie und pathologischen Anatomie der verschiedenen Idiotieformen: II. Tuberöse Sklerose, Monatschr. f. Psychiat. u. Neurol. **24**:106-150, 1908.

3. Critchley, M., and Earl, C. J. C.: Tuberose Sclerosis and Allied Conditions, Brain **55**:311-346 (Sept.) 1932.

4. Nitsch, M.: Augenhintersgrundsbefund bei tuberöser Hirnsklerose, Ztschr. f. Augenh. **62**:73-75, 1927.

5. Kuchenmeister, E.: Ueber einen Fall von Pringlescher Krankheit mit Veränderungen am Augenhintergrund und an den Schleimhäuten von Blase und Mastdarm, Dermat. Wchnschr. **99**:133-137, 1934.

6. Bau-Prussakakowa, S.: Ueber einen Fall von tuberöser Hirnsklerose mit Netzhautveränderungen und benignem Verlauf, Ztschr. f. d. ges. Neurol. u. Psychiat. **145**:275-282, 1933.

and F. Rintelen [7] such tumors of the disk are described. G. Salom [8] reported the ocular findings for twenty-nine persons in five families, in each of which there was at least one case of well defined tuberous sclerosis. He listed four types of retinal changes: (1) yellowish red spots; (2) blurred pigmented spots; (3) nevoid spots and nevi, clearly outlined, and (4) pigmented changes identical with those of pigmented retinitis. Probably only the first of these represents tumor. Von Herrenschwand [9] reported a case in which there were tumors near the margins of each papilla, describing one of them as resembling a "mulberry composed of glittering white nodules." A. Vogt [10] described a retinal tumor resembling a mulberry. Overbosch [11] reported a case in which there were "bilateral typical tumors of the fundus oculi." Guillain and

Accepted Cases of Retinal Tumors in Tuberous Sclerosis

Author	Year	Number of Cases	Tumor of the Disk	Pathologic Study Made
van der Hoeve [1]	1921	6	1 case	2 cases
Schob [15]	1925	1	No	Yes
Overbosch [11]	1926	1	No	No
Nitsch [4]	1927	1	Yes	No
von Herrenschwand [9]	1929	1	No	No
Feriz [16]	1930	1	No	Yes
Salom [8]	1932	5	No	No
		(2 doubtful)		
Critchley and Earl [3]	1932	1	No	No
Bau-Prussakakowa [6]	1933	1	Yes	No
Kuchenmeister [5]	1934	1	Yes	Yes(1935)
Guillain and Lagrange [12]	1934	1	No	No
Vogt [10]	1934	1	No	No
Gottlieb, J. S., and Lavine, G. R.: Arch. Neurol. & Psychiat. **33**:379-388 (Feb.) 1935	1935	1	No	No
Rintelen [7]	1935	1	Yes	No
Storchhein, F., and Taube, E. L.: Am. J. Ophth. **19**:508-509 (June) 1936	1936	1	No	No
Total		24	5	5

Lagrange [12] described a case in which there were choked disk in each eye and, in one eye, the white spots typical of van der Hoeve's "phakomatosis."

7. Rintelen, F.: Fundusveränderungen bei tuberöser Hirnsklerose, Ztschr. f. Augenh. **88**:15-19 (Dec.) 1935.

8. Salom, G.: Contributo allo studio sulla familiaritá della sclerosi tuberosa, Rassegna di studi psichiat. **2**:945-960, 1932.

9. von Herrenschwand, F.: Ueber Augenhintergrundveränderungen bei tuberöser Hirnsklerose, Klin. Monatsbl. f. Augenh. **83**:732-736, 1929.

10. Vogt, A.: Seltener Maulbeertumor der Retina bei tuberöser Hirnsklerose, 9 Jahreverfolgt, Ztschr. f. Augenh. **84**:18, 1934.

11. Overbosch, J. F. A.: Vertooning van een patient met tubereuse sclerosis, Nederl. tijdschr. v. geneesk. **2**:2632-2635, 1926.

12. Guillain, G., and Lagrange, H.: Phacomatose rétinienne de Van der Hoeve dans un cas de sclérose tubéreuse, Bull. et mém. Soc. méd. d. hôp. de Paris **50**: 1421-1425 (Nov. 12) 1934.

In tuberous sclerosis, besides the typical, multiple small tumors in the cortex of the brain, there are usually tumors projecting more or less into the ventricles; it is these which sometimes cause choked disk. Nystagmus, strabismus and myopia have rarely been found.

From the descriptions in some of the cases of tuberous sclerosis reported it is difficult to say exactly how many patients have shown retinal tumors, but we have found twenty-four cases in which we think they were present.

The description of van der Hoeve [1] remains as the pattern for an investigation of a case of this condition.

Fig. 1.—Appearance of the patient in 1931, after he had received ten treatments (coagulation) on the left side of the face and nose. The typical appearance of adenoma sebaceum is still seen on the right.

REPORT OF CASE

An Italian man aged 20 was admitted to the neurologic service of the Rhode Island Hospital under the care of Dr. William N. Hughes on March 17, 1935, because of frequent, severe convulsive attacks.

At the age of 4 years he began to have mild convulsive seizures, which were infrequent until about two years before admission to the hospital, since when they had increased in frequency and severity. At the age of 14 he was seen by Dr. F. Ronchese, of Providence, R. I., because of a nodular eruption on his face. A diagnosis of adenoma sebaceum was made and was confirmed by biopsy. The urine, the blood count and the blood chemistry were normal. The Wassermann reaction was negative. Psychometric tests showed the patient to be four years

retarded. He was presented at the Atlantic Dermatological Conference, held in Boston on Nov. 10, 1930, and the case was reported in print in 1931.[13]

Two weeks before admission, after three months in which there were no attacks, he had a very severe attack, followed by two others. On the day before admission he had fourteen.

He was found to have bronchopneumonia. On March 18 he was free from convulsions and appeared normal mentally. An extensive neurologic examination gave negative results except for weakness of the lower part of the right side of the face. He did not appear mentally defective. On both sides of the neck were some small pigmented, fibrous cutaneous tumors.

The right pupil was larger than the left; both reacted to light. Vision in the right eye was reduced to counting of fingers at 2 feet (60 cm.). Vision in the left eye was nearly normal. There was no gross refractive error. The right disk was obscured by a white, shiny mass with a nodular surface, which extended forward 5 diopters, overlying the vessels.

On the next day the patient had many convulsions and died.

On later investigation the patient's father was found to have adenoma sebaceum of the face (proved by biopsy) and to suffer from peculiar attacks which were not typically epileptic. Ophthalmoscopic examination of his eyes revealed no retinal tumors.

PATHOLOGIC FEATURES

In 1923 van der Hoeve[14] published a pathologic study of the retinal tumors from two of his six patients. From one he had only a single eye, which was enucleated because of a suspicion of malignant change. In this eye there were a large tumor of the optic disk, which he regarded as the primary growth, and several smaller secondary tumors scattered over the retina. In the second case both eyes were removed at autopsy. There were no tumors of the papilla, but there were several retinal tumors in each eye. The histologic picture in the two cases were essentially alike. He described the tumors as being made up of nerve fibers and a peculiar kind of cell. He expressed the belief that the fibers came from the nerve fiber layer and extended through holes in the membrana limitans interna into the overlying tumor. The cells were described as having much cytoplasm, which in places fused with that of neighboring cells to form a syncytium. The nucleus was usually large, with a prominent nucleolus. In the papillary tumor there was a large incrustation. In all the tumors there were spaces without any special lining, filled with blood and serum. Blood vessels were sparse. The tumors appeared to originate in the nerve fiber layer and frequently extended to involve the ganglion cell layer but seldom any others. On the surface of the large tumor of the optic disk were button-like projections which, he believed, became pinched off, to float in the humor, and then became

13. Ronchese, F.: Nevus Fibro-Angiomatosus (Adenoma Sebaceum Pringle). Arch. Dermat. & Syph. **23**:753 (April) 1931.

14. van der Hoeve, J.: Augengeschwülste bei der tuberösen Hirnsklerose (Bourneville) und verwandten Krankheiten. Arch. f. Ophth. **111**:1-16, 1923.

implanted elsewhere on the retina. Indeed, he actually observed this to occur, through the ophthalmoscope.

We have been able to find only three additional reports of pathologic studies of these tumors—all in German.

In 1925 Schob [15] published his observations on the eyes of a 6 year old child who died of tuberous sclerosis. There were no tumors of the disk. Grossly, small, whitish, slightly raised areas could be seen on each retina. Microscopically, some were flat and some sharply raised. He described buds and daughter buds with constricted necks, on the surface of the larger tumors. In his case the tumors were limited to the nerve fiber layer. He, too, described fibers but concluded that they were definitely glial fibers and not nerve fibers. He, too, described a syncytium, and giant cells with two or three nuclei. The nuclei were large and vesicular, with little chromatin and with from one to three nucleoli. They were oval, long, curved and sometimes dumb-bell shaped.

In 1930 Feriz [16] reported a histologic study of a solitary tumor of the left eye which did not involve the disk. This tumor involved the nerve fiber layer and the ganglion cell layer. He described large "spider and star-shaped" cells. The cytoplasm stained strongly eosinophilic. In places the walls of the cells were indistinct, suggesting a syncytium. Many cells contained vacuoles. The nuclei were round to three cornered. There were bands and whorls of fibers which he considered "neuroglia-like."

The third such study was by Kuchenmeister [17] in 1935. Both eyes were involved. The optic disks were not involved. Unlike the observations in previous reports, all the layers were invaded. In places there was calcification, and in the choroid there was some bone formation. The cells varied in size and shape. There were "new-formed glial fibers."

At postmortem examination in our case, typical lesions of tuberous sclerosis were seen, including multiple tumors of the brain, rhabdomyoma of the heart, lipofibromas of the kidneys and adenoma sebaceum of the Pringle type. In the right eye was a single raised, whitish tumor 3 mm. in diameter, which covered the upper and inner two thirds of the optic disk.

Histologic preparations showed the tumor overlying the optic disk and, as in Kuchenmeister's case, involving all the layers of the retina. It also invaded a portion of the optic nerve. In the central portion of

15. Schob, F.: Beitrag zur Kenntnis der Netzhauttumoren bei tuberöser Sklerose, Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:731-740. 1925.

16. Feriz, Hans: Ein Beitrag zur Histopathologie der tuberösen Sklerose. Virchows Arch. f. path. Anat. **278**:690, 1930.

17. Kuchenmeister, E., cited by Fleischer: Ueber klinischen und anatomischen Befund bei tuberöser Hirnsklerose, Ztschr. f. Augenh. **88**:158 (Jan.) 1935.

the tumor was a large, irregular mass of ossification, and about this were calcium-containing concretions. Van der Hoeve mentioned a "large incrustation," and Kuchenmeister mentioned "calcification and bone formation." The surface of the tumor was smooth; there were no buds, as were described by van der Hoeve and Schob. At the periphery of the tumor a thin layer of tumor cells extended out over the surface of the external limiting membrane.
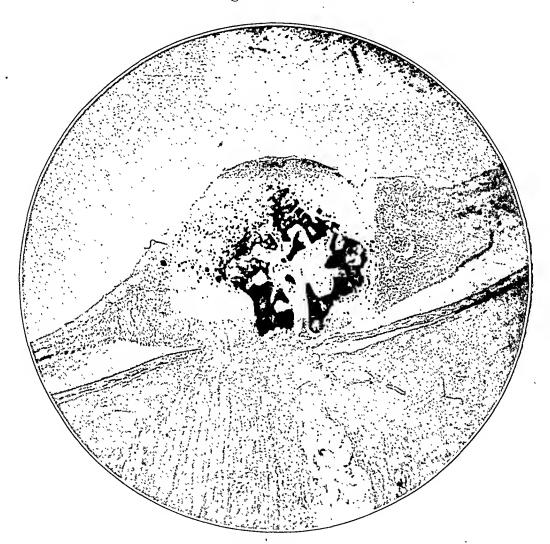
Fig. 2.—Low power photomicrograph of a section through the nerve head, showing the location and extent of the tumor. The ossification in the center of the tumor is prominent. The thin dark line on the surface of the retina peripheral to the main tumor is an extension of tumor cells.

The cells varied greatly in size and shape. Some were almost round. Others were markedly angulated. The boundaries of the cells were often indistinct, suggesting a syncytium. Many cells were elongated, and the cytoplasm was drawn out to a thick process.

The nuclei were round or oval and vesicular. A single nucleolus was frequently prominent. Multinucleated cells were seen, having from two to five nuclei.

We were at first unable to demonstrate any production of glia fibrils, but in preparations stained for us with phosphotungstic acid by Dr. J. G. Greenfield, of the National Hospital for Nervous Diseases, London, there could be seen short, fine fibrils in the processes of the larger cells.

The type of cell was not definitely determined. Van der Hoeve [14] suggested that these large cells are descendants of the first anlage of the retina and that they are neurocytes which have not differentiated
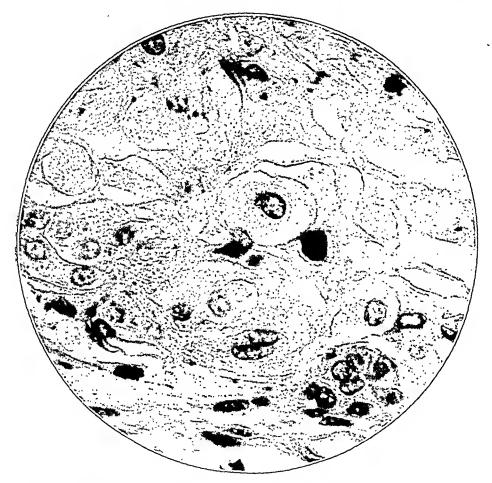
Fig. 3.—High power photomicrograph of tumor cells, showing variation in size and shape, vacuolation and the tendency to form a syncytium.

into glia or ganglion cells or "glia-neurocytes." In the belief that they arise from embryonic cell rests, he proposed that they be called "phakomata," from the Greek word *phakos,* meaning mother-spot. Combining these words, he arrived at the term "neurocyto-phakomata retinae or papillae."

Schob [15] concluded that these tumors are closely related to other gliomatous tumors and that they take origin from the nerve fiber layer, which normally contains glia cells. He proposed no definite name.

Feriz [16] considered these tumors much like those seen in the brain and expressed the belief that they are made up of atypical neuroglia-like tissue.

Because of our ignorance concerning the embryology of the eye, we sought the help of Miss Ida Mann, of London, England, whom we quote:

With regard to the derivation of the cells, I think, as they are glial, one will have to postulate their descent from the first stage (from the fourth to the fifth
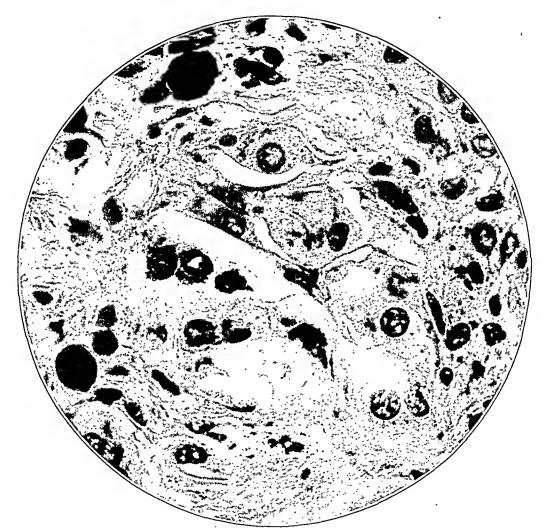
Fig. 4.—A group of tumor cells with blunt protoplasmic projections suggesting nerve cells, and a multinucleated syncytial mass. The rounded black masses are calcium-containing concretions.

week) of retinal differentiation, when the cells destined to form supporting structures are separating from the primitive neuro-epithelium. I should consider that the cells in your tumor . . . have begun to differentiate into the specialized glia of the retina, but have not got very far. . . . There are also in your tumor some cells in which fibrils cannot be seen. These may be neurocytes, but if they are they are probably derived from the inner neuroblastic layer, since these cells begin at the same time as the müllerian fibers. I should say that the tumor arose during stage 2 of retinal differentiation (from the sixth week to the third

month) from glia cells of the inner neuroblastic layer, with possibly a few undifferentiated ganglion and amacrine cells included. The fact that it is on the disk need not cause confusion, since the primitive epithelial papilla, before it is replaced by nerve fibers, is well known to be able to differentiate into abortive retina. The fact that the tumor is on the disk is itself in favor of its having arisen about the 15 mm. stage to the 20 mm. stage. If it had arisen earlier, the fetal fissure would have been open, and one would expect its closure to have been interfered with by the tumor at its upper end and some sort of coloboma of the disk to have resulted. If, on the other hand, it had arisen later, after nerve fibers had replaced
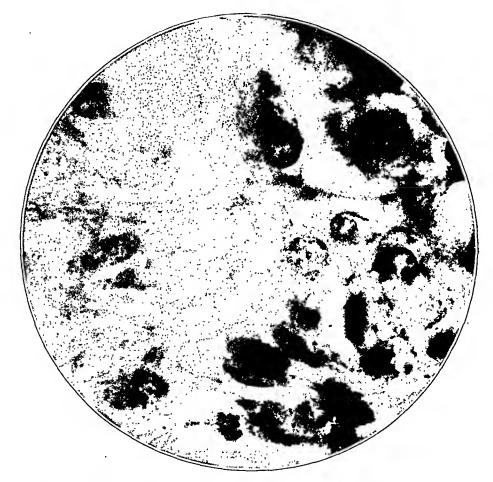
Fig. 5.—Section showing a few fibrils within the cytoplasm of some cells; Mallory's phosphotungstic acid-hematoxylin stain.

the neuro-epithelial cells (at the 25 mm. stage and after) it would be difficult to see where its cells of origin came from, since Bergmeister's papilla is definitely glial by then, and dedifferentiation of cells, once finished, is hardly to be postulated in this case.

It seems evident that these tumors must arise from some early cell of the embryonic retina. It appears definite that glia fibers were present in the cases of Schob, Feriz and Kuchenmeister. None were present

in van der Hoeve's case. Van der Hoeve described nerve fibers which were not seen in any other case.

Grinker [18] suggested that "these tumors are probably astroblastic *neoplasms of low growth possibilities but as yet enough has not been* reported to be sure of classification."

It seems, from a review of the descriptions in the literature, from the observation in our case and from a consideration of the embryology, that these growths are largely made up of glia cells. Together with the lesions in the brain and other organs, they probably should be considered as hamartomas rather than true tumors.

SUMMARY

Twenty-four instances of retinal tumor in cases of tuberous sclerosis are collected from the literature. In only four of the studies reported was a pathologic examination made. We have added a fifth such study, the first to be recorded in this country. The case reported by us is the sixth in which there was a tumor of the optic disk. We feel that cytologic evidence, together with the embryologic possibilities, justifies the conclusion that these tumors are essentially gliomatous.

We think it probable that if psychiatrists, ophthalmologists and pathologists have this lesion in mind the incidence will prove to be much greater than the meager literature suggests.

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18. Grinker, Roy R., in Penfield, Wilder: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, p. 1058.

SECONDARY CATARACT

WITH PARTICULAR REFERENCE TO TRANSPARENT GLOBULAR BODIES

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Transparent globular bodies frequently constitute an interesting feature of after-cataract. A few of the many bodies of this nature observed by us, as seen with the slit lamp and the corneal microscope, are illustrated in figures 1, 2, 3, 4, 5 and 6.

Little has been written about this peculiar and frequently occurring phenomenon, although J. Hirschberg [1] remarked on "drops" which not only form a part of various cataracts but are found in after-cataract membranes. Elschnig [2] described them exactly and made microscopic examinations. He failed to mention Hirschberg.

Koby [3] stated that transparent globules are found more frequently in the young than in the old and that he rarely met with globular cells after the fiftieth year. This has not been our experience. There seems to be no preference in regard to age. Of the four cases of after-cataract with formation of globules described by Vogt [4] three were those of patients aged 62, 63 and 64 years, respectively.

The globules are seen only after division of the capsule of the lens, never after all the remains of the capsule have been removed. Visual

1. Hirschberg, J.: Einführung in die Augenheilkunde, Leipzig, Georg Thieme, 1901, p. 159; quoted by Vogt, A.: Lehrbuch und Atlas der Spaltlampenmikroskopie des lebenden Auges, Berlin, Julius Springer, 1931, p. 634.

2. Elschnig, A.: Klinisch-anatomischer Beitrag zur Kenntnis des Nachstares, Klin. Monatsbl. f. Augenh. 49:444, 1911.

3. Koby, F.: Slit-Lamp Microscopy of the Living Eye, ed. 2, translated by Charles Goulden and Clara Lomas Harris, Philadelphia, P. Blakiston's Son & Co., 1925, p. 282.

4. Vogt, A.: Lehrbuch und Atlas der Spaltlampenmikroskopie des lebenden Auges, Berlin, Julius Springer, 1931, pp. 716, 717 and 719, figs. 1450 a, b and c and 1459 b, c and d.

acuity is affected, depending on the extent of the occupation of the pupillary area by the globules. They vary in size up to 2 mm. in diameter (fig. 1) and are similar in appearance and action to perfectly transparent soap bubbles. Sometimes one or more can be seen hanging
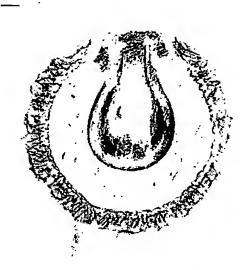
Fig. 1.—Transparent globular body seen in the eye of Mrs. F. K., aged 65, who had diabetes and arteriosclerosis. The drawing was made eight days after extraction of the lens. Six weeks later the large globule could not be seen, but there was a bunch of small globules in the upper pupillary space. These continued to change until the last time the patient was seen, thirty-two months after the operation.
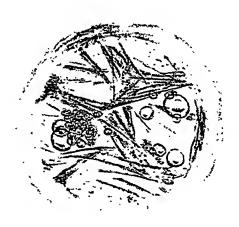
Fig. 2.—Transparent globular bodies seen in the eye of Miss S. H., aged 25. The drawing was made two years after discission for congenital cataract.

by a thin pedicle, but mostly they occur in bunches. When the globules are very small, the mass appears like foam or froth. Not infrequently we have seen the whole pupil filled with them.

A   B
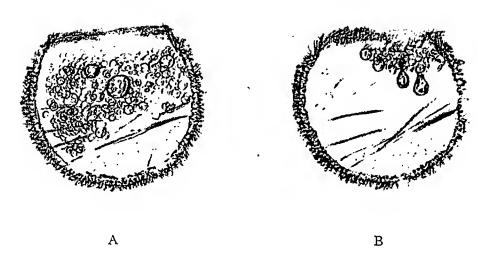
Fig. 3.—*A*, transparent globular bodies seen in the eye of Mrs. E. M., aged 58. The drawing was made two years after combined extraction of senile cataract. There was hernia of the vitreous below the lower edge of the remaining portion of the capsule. Vision was 6/20 with correction. The condition continually changed. *B*, appearance of the same eye two years later; the condition was still changing.

A   B
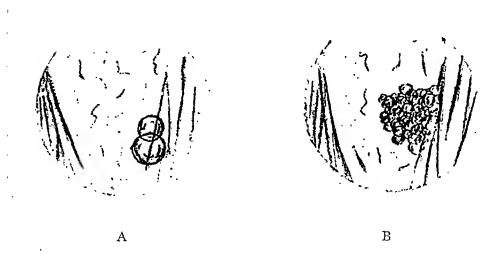
Fig. 4.—*A*, transparent globular bodies seen in the eye of J. D., aged 8. The drawing was made seventy days after linear extraction and iridectomy for traumatic cataract. Ten days previously only one globule was seen. *B*, appearance of the same eye one month later. The globules had increased in number, and four months after operation the pupil was entirely filled with them.

Whether a single globule or a large number are seen, they always extend from a portion of the remaining part of the capsule of the lens. Some are spherical; others are ellipsoid. Often they fluctuate with the movements of the eye. They change continually in appearance.
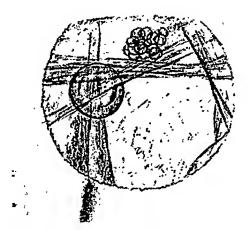
Fig. 5.—Transparent globular bodies seen in the eye of Mrs. M., aged 63. The drawing was made two years after combined operation for senile cataract. When the patient was seen three months later, the large globule had disappeared.

Fig. 6.—Transparent globular bodies seen in the eye of A. B., aged 11, eight days after unsuccessful discission for congenital dislocated cataract. The knife needle merely nicked the capsule of the lens, at which place the bunch of globules grew.

From time to time the picture differs. In a surprisingly short time some globules disappear and new ones appear. They are produced rapidly, are seen a few days after operation and continue to form, vanish and form again, the process continuing for years. One is given the impression that they blow up and finally burst like a toy balloon. Sometimes, when the process is arrested, the wall of the globule gradually loses its transparency.

While the exact nature of these bodies is not understood and while they are seen more frequently after operation, they may occur after any separation of the capsule, as in the case (fig. 6) in which they were produced after a knife prick of the capsule. Vogt[5] has seen them develop in the interior of the lens after contusion.

In one instance, that of a woman 58 years of age, the whole pupillary space was filled with a layer of these globules. The entire membrane was removed by the method of Lyster.[6] The microscopic appearance is illustrated in figures 7 to 11, inclusive. The gross specimen consisted of a small, irregularly quadrilateral grayish membrane. After fixation in a 10 per cent dilution of solution of formaldehyde U. S. P., it was transferred to a 70 per cent solution of alcohol and examined under glass with the slit lamp. When examined in vivo this tissue had
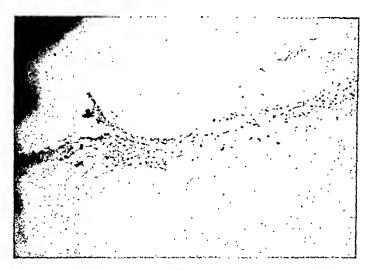
Fig. 7.—Section through the membrane of the secondary cataract, showing the anterior capsule of the lens and proliferated capsular epithelium.

exhibited innumerable small glistening rounded globules, but after fixation the greater number of these could not be seen. There was, however, near one margin of the material a small area in which there were still a few globules. These globules were small but were typical of those seen in the eye previous to extraction of the secondary membrane. Therefore, it was considered advisable to section this membrane serially.

A typical section of the membrane formed an arc of tissue about 6 mm. in length, consisting chiefly of the posterior capsule of the lens in the middle portion and the equatorial and the anterior capsule

5. Vogt,[4] fig. 1330.

6. Lyster, Theodore C.: Management of Dense Secondary Cataract, Am. J. Ophth. **16**:122 (Feb.) 1933.

of the lens at each end. In many of the sections the anterior capsule was folded inward, and there was included in the space so formed a number of degenerated lens fibers. The posterior capsule of the lens was thin and presented no noteworthy features except that in several
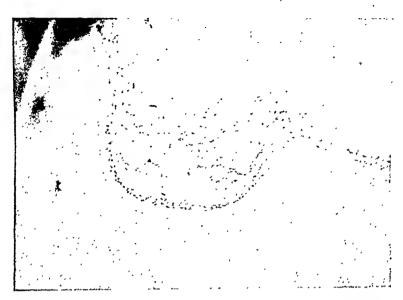
Fig. 8.—Fold of the anterior capsule of the lens, within which are seen large bladder cells and proliferated capsular epithelium with adjacent, interlacing hyaline bands.
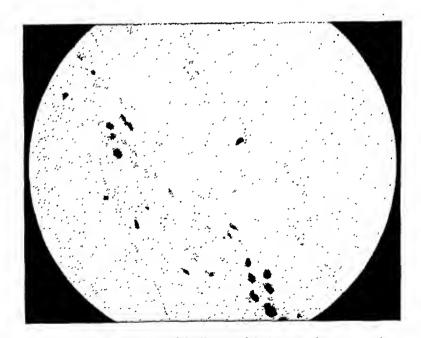
Fig. 9.—Proliferated capsular epithelium with loops of apparently newly formed capsular tissue.

sections there was a longitudinal split and in others the anterior capsule, which was moderately thick, showed capsular epithelial cells on both sides. This appearance must have resulted from proliferation of the

capsular epithelium around the torn edge of the anterior capsule. At times these cells exhibited marked changes. The cell wall was circular; the cytoplasm had disappeared, except for a few granules, and the nucleus either was flattened against the wall or was entirely absent. At other points there was marked proliferation of capsular epithelium, so
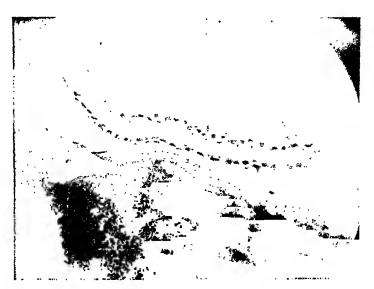
Fig. 10.—Capsular epithelium which had proliferated to cover both sides of the anterior capsule.
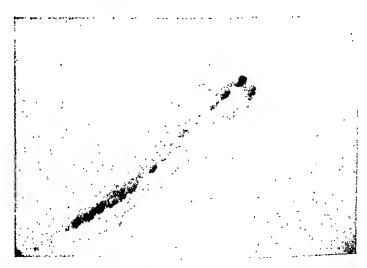
Fig. 11.—Two vesicle-like capsular epithelial cells. They appear different from bladder cells.

that the cells were four or five layers in depth. In the places in which there were such layers of epithelial cells they were not piled on top of one another but were separated by loose interlacing bands of hyalin-like tissue similar to that of lens capsule. These hyalin-like bands

were in a position to be the product of an abnormal activity of the
epithelial cells. In some places cross-sections of lens fibers had the
appearance of large, irregularly rounded spaces filled with a fine
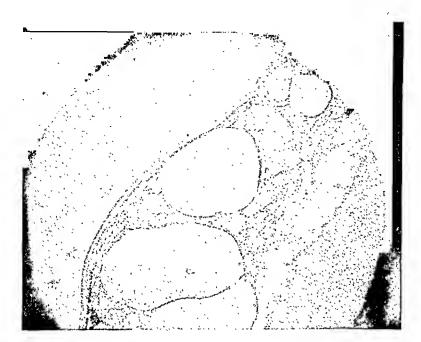
Fig. 12.—Vacuoles within a lens dislocated into the vitreous. The capsule of
the lens was intact.

Fig. 13.—Vesicle-like formations beneath the capsule of a lens dislocated into
the vitreous.

granular deposit—the so-called bladder cells. There were no spheres
of Morgagni. No calcareous areas were noted, and no slits which might
have contained cholesterol crystals were present.

The three features which are pertinent to the present discussion are: first, the vesicular or bladder cells; second, the interlacing bands of hyaline tissue, and, third, the cystic appearance of a certain number of the proliferated and swollen capsular epithelial cells. The bladder cells
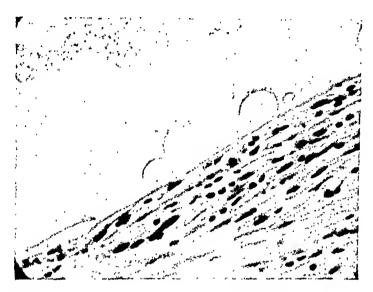
Fig. 14.—Group of vesicles on the surface of a degenerated retina.
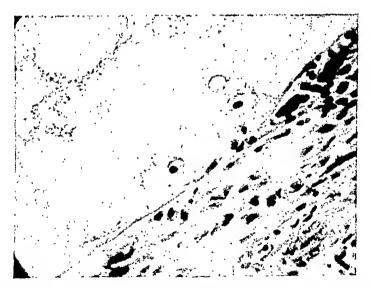
Fig. 15.—Group of vesicles, apparently derived from cells on the surface of a degenerated retina.

probably do not account for the appearance of the globules described by Hirschberg and by Elschnig. These cells commonly are found within and should be seen in most cataractous lenses. But no such formations were noted in the cases which we observed. Concerning the second feature, the interlacing hyaline bands, these had a folded appearance at

places that suggested collapsed vesicles, but it seems unlikely that they could account for the delicate walled structures under consideration. It seems most likely, therefore, that the globules of Hirschberg and Elschnig are formed from cystically enlarged capsular epithelial cells.

Clinically, it had been found that the occurrence of these globules on membranes of secondary cataract was so frequent that it was thought advisable to examine sections of enucleated eyes containing secondary cataracts in order to determine whether any structures other than those found in our specimen could account for the formations seen by the slit lamp. Accordingly, a number of specimens from the collection of pathologic material at the University of Pennsylvania and at the Wills Hospital were examined. No additional information was obtained from this search.
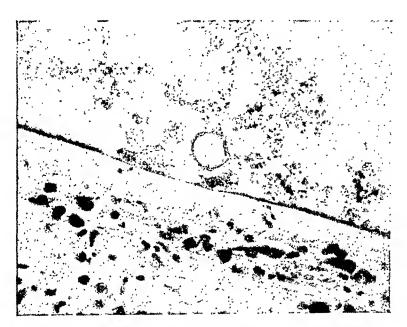
Fig. 16.—Vesicle on the surface of a degenerated retina, in which a flattened nucleus is visible, indicating the cellular origin of the vesicle.

The formation by intra-ocular cells of a large thin-walled vesicle-like structure is not limited to the capsular epithelium of a secondary cataract. Such a vesicle-like structure was seen in a lens dislocated within the vitreous but otherwise intact. The capsular epithelium of this lens showed that it was abnormal to a certain extent, in that it had proliferated so as to line the whole posterior capsule. This structure is seen in figures 12 and 13. It is also known that similar processes occur in cells on the surface of degenerated retinas, as shown in figures 14 and 15. The cellular derivation of such a vesicle is clearly indicated in figure 16, in which a flattened nucleus is still visible.

It is noteworthy that in the observations described by Elschnig the histologic examinations were made on the eyes of two patients.

In the report of neither case is there an accompanying description of the results of clinical or slit lamp examination. The histologic changes were essentially those of a secondary membranous cataract with a well marked Soemmering ring. The hemispherical nodules which he described were solid formations, part of which contained nuclei and part of which did not. He stated that the red-stained ones were without a nucleus. This indicates to us that they had undergone greater degeneration. He did not distinguish these sharply from the well known bladder cells.

A SKETCH OF THE EARLY DAYS OF OPHTHALMOLOGY IN PHILADELPHIA

BURTON CHANCE, M.D.

PHILADELPHIA

Ophthalmology—the first of the medical specialties—owes much to the practitioners of Philadelphia. It took many years, however, before in this city the "specialty" reached a status sufficiently secure to receive the dignified support of the medical profession, as well as the respect of the community, enabling it to go on and on to maintain, nationally and internationally, its place in the science of medicine.

Until after the middle of the eighteenth century in this country, just as in England and on the Continent, diseased eyes were treated by itinerant quacks, or by those who, by self-teaching, from their own experience or from what they had learned somewhere in Europe acquired more or less skilful ability to relieve sufferers from ocular disease. Such persons were known as "oculists," to be regarded with suspicion and aversion by the regular physicians, both in this country and in England, so that until about the second quarter of the nineteenth century the title oculist was one of opprobrium.

Nevertheless, here and there certain systematically educated surgeons deliberately undertook to treat ocular conditions. They operated for squint and for cataract, incised abscesses of the lacrimal sac and attempted the repair of eyelids torn and distorted in consequence of wounds received in warfare and from accidents in the early industries. Soon after the University of Pennsylvania School of Medicine began to graduate students, young physicians sought further education in London, Edinburgh, Paris and Vienna. Their nebulous knowledge of the anatomy of the orbit and its contents became more exact, and after returning to America they confidently and deliberately cared for a greater variety of disorders.

Certain practitioners, fortified by the increased knowledge gained by their experience in the Revolutionary army and with greater understanding of their problems, entered into civil practice after the war; yet, a "medical man" in those days was compelled to be all things to all men—surgeon, physician, obstetrician and nurse. Preeminent in

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Philadelphia at the end of the eighteenth century and in the first quarter of the nineteenth was Philip Syng Physick, deferentially spoken of as "the father of American surgery." He was distinguished for his operation on the crystalline lens and famous as an "extractor of cataract." Another pioneer in ophthalmology was Gibson, who devised an operation on the straight muscles of the eye for the correction of squint.

Dr. Physick, a native of Philadelphia, after finishing his course in arts at the University of Pennsylvania, became the student of Dr. Adam Kuhn, with whom he continued until 1788. Then he went to London,

PHILIP SYNG PHYSICK M.D.

Fig. 1.—Philip Syng Physick in early years.

where he so attracted John Hunter that he was invited to become a member of that great master's household. Through Hunter's influence Physick secured a position on the house staff of St. George's Hospital, where he was afforded many opportunities to acquire extensive surgical proficiency. After a few months with Hunter he went to Edinburgh to graduate in medicine at the university of Edinburgh, which for many years was the source of American medical education. He returned to America in 1792, being then only 24 years of age, and began practice

in Philadelphia, which he conducted with distinction for forty years, throughout which time numerous cases of ocular disorders came under his observation. He was appointed surgeon to the Pennsylvania Hospital in 1794 and served in this capacity for many years. Some of his pathologic specimens preserved in the museum are still in good condition, and when certain of them were studied in recent years the diagnoses originally entered were confirmed by the histologic observations. Physick wrote little, and his records are scanty; yet he kept

Fig. 2.—Two pages from "Notes on Surgery," taken from the lectures of Philip Syng Physick, M.D., professor of surgery in the University of Pennsylvania, by William Elmer, a student in medicine, during the winters of 1808, 1809 and 1810. The notebook is in the library of Dr. Walter G. Elmer, of Philadelphia.

a private journal of his interesting cases. The first recorded relates to the blindness of a woman which he relieved by the extraction of the opaque lens. Couching was the usual operation of that day, but Physick had discarded that method because of defects inherent in it. Instead, he adopted the method of Baron Wenzel, and was highly successful in using it. Wenzel, as is known, incised the cornea at the upper limbus with

a long blade, the edges of which sloped, forming an isosceles triangle.
The blade, as it passed across the expanse of the aqueous chamber,
plugged the incision, preventing extrusion of the iris and thus made a
clean wound through which the unbroken lens could pass. No iridec-
tomy was done. The capsule was lacerated with a hooklike point, and
the lens was withdrawn with another hook—a true extraction. Although
Physick succeeded well in the wider fields of surgery, it is likely that

Engraved by C. Goodman & R. Piggot from an original Picture by Sully

.J.S.DORSEY.M.D.

Fig. 3.—John Syng Dorsey.

his popular fame rested not a little on his skill in ophthalmic surgery. It
is of interest to note that the last operation he performed, which took
place in the midsummer of 1837 (when he was in feeble health), was
on the eye. He was famous as a teacher, and his favorite nephew, Dr.
John Syng Dorsey, who assisted him, endeavored to carry out and

continue his methods, thus extending Physick's teachings, which he compiled in his "Elements of Surgery." This served as a surgical text-book for many years. But this admirable association was terminated by the untimely death of Dr. Dorsey in 1818, at the age of 35 years.

ELEMENTS

OF

SURGERY

FOR THE USE OF STUDENTS

WITH PLATES

by

JOHN SYNG DORSEY M.D.

ADJUNCT PROFESSOR OF SURGERY IN THE UNIVERSITY OF PENNSYLVANIA —

ONE OF THE SURGEONS OF THE PENNSYLVANIA HOSPITAL &

VOL.I.

—————, for want of timely care
Millions have died of medicable wounds.

Armstrong.

J. Ellwes. Sc.

PHILADELPHIA.

Published by Edward Parker & Kimber & Conrad

Fig. 4.—Title page of Dorsey's "Elements of Surgery."

Physick's manner as a teacher and clinician must have been impressive. The University of Pennsylvania possesses several manuscript volumes of notes made by his students and assistants. His courses were entirely clinical and covered all subjects relating to the surgery of his day. I had the great privilege of seeing the notes made by a Dr. Elmer in three years about 1810. The chapters on diseases of the eye are

extensive and give with unusual detail Physick's treatment of cataract. The notes of Elmer are explicit in all details. In that day, smallpox having played its devastating part, there were many cases of occluded pupils and leukomatous corneas. Physick had much success in his efforts to form an artificial pupil, for which he invented forceps with a punchlike beak with which to perforate the membrane of the iris.

Fig. 5.—A page from "Notes from Medical and Surgical Cases" taken from 1801 to 1811 by John Syng Dorsey. The notes shown were taken in 1810.

William Gibson, too, another graduate of the University of Pennsylvania, was especially interested in diseases of the eye. He has been credited with being the first in America to operate for convergent strabismus. His procedure, which he described in 1818, antedated that by Dieffenbach by many years. He began practice in Baltimore, and on the retirement of Physick was called in 1819 to succeed Physick at the University of Pennsylvania, a position which he held until 1855.

Gibson wrote a textbook entitled "The Institutes and Practice of Surgery," published in 1824, in the second volume of which he presented in the successive eight editions a résumé of the state of ophthalmic practice as it existed in the first and second quarters of the nineteenth century. Gibson dedicated his textbook to Physick, and from it most American surgeons secured their basic education in surgery during a

Fig. 6.—Two pages from "Notes from Cases in Surgery" (these cases were observed in the practice of Dr. Physick), taken by John Syng Dorsey in 1798-1799.

period of thirty years. Gibson continued to be prominent in American ophthalmology for many years, doing much toward the successful treatment of cataract, having perfected a linear extraction as early as 1811; yet one seldom hears of him today!

At the opening of the session at the University of Pennsylvania for the academic year 1793-1794 there was enrolled one Elisha North.

destined to inaugurate a distinct epoch in the practice; indeed, he became a pioneer of ophthalmology in America. North, who had never received a diploma, assisted his father, a self-trained country practitioner in Goshen, Conn. He secured an apprenticeship to a prominent physician in Hartford, Conn., and later came to Philadelphia because of the

Fig. 7.—A page from Philip Syng Physick's lectures on surgery for November 1807.

celebrity of the teachers at the University of Pennsylvania. On returning to New England in 1812, he settled in New London, Conn., and decided to devote himself to diseases of the eye, for he had doubtless received much inspiration from his teachers at the University of Pennsylvania. In 1817 he opened an infirmary for patients with diseases

of the eye, the first in this country. That experiment, distant though its seat was from Philadelphia, was not without effect on the practice in this city, for because of it the attention of the medical profession was focused on the necessity for greater concern about diseases of the eye. Hitherto the majority of the practitioners were entirely without comprehension of the seriousness of ocular diseases and consequently

Fig. 8.—William Gibson. This photograph was taken from the portrait on wood by Joseph Wood, in the possession of the College of Physicians of Philadelphia.

had only a faint conception of the importance of the relation they bore to general medicine. North became the most distinguished practitioner of Connecticut, but he did not continue in ophthalmology.

There was no special work devoted to the diseases of the eye available in English for the guidance of early practitioners until the opening of the nineteenth century. No systematized presentation of the

pathology of the eye existed in any language until James Wardrop, a distinguished surgeon in London, issued his treatise in 1808. Wardrop was the author of the infamous and offensive "Intercepted Letters," published in the early issues of the *Lancet,* in certain of which, despite his own skill in ophthalmic surgery, he violently opposed the establishment of an ophthalmic hospital in London.

GEORGE FRICK

1793-1870.

Fig. 9.—George Frick.

The first work on ophthalmology published in America was "A Treatise on the Diseases of the Eye; Including the Doctrines and Practice of the Most Modern Surgeons, and Particularly Those of Professor Beer," written by George Frick, a native of Baltimore. After receiving his degree at the University of Pennsylvania, in 1815, Frick went to Vienna to study under Dr. George Joseph Beer, at that day the foremost ophthalmologist in Europe. Beer had been one of the

two students chosen by Joseph Barth, celebrated as the first "Professor" of ophthalmology in medical history.

Barth was an uneducated and illiterate man who had acquired extraordinary facility in operating for cataract; he had learned how to perform this operation from watching the celebrated Baron Wenzel.

A

TREATISE

ON THE

DISEASES OF THE EYE;

INCLUDING THE

Doctrines and Practice

OF THE

MOST EMINENT MODERN SURGEONS,

AND PARTICULARLY THOSE OF

PROFESSOR BEER.

BY GEORGE FRICK, M. D.

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Ophthalmic Surgeon to the Baltimore General Dispensary.

WITH AN ENGRAVING.

Baltimore:

PUBLISHED BY FIELDING LUCAS, JUN.

JOHN D. TOY, PRINTER.

1823.

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A NEW EDITION WITH NOTES,

BY RICHARD WELBANK,

MEMBER OF THE ROYAL COLLEGE OF SURGEONS, AND OF THE MEDICAL AND CHIRURGICAL SOCIETY OF LONDON.

With an Engraving.

LONDON

PRINTED FOR JOHN ANDERSON,

MEDICAL BOOKSELLER, WEST SMITHFIELD;

ADAM BLACK, EDINBURGH; W. R. M'PHUN, GLASGOW; AND HODGES AND M'ARTHUR, DUBLIN.

1826.

Fig. 10.—Title page of Frick's "A Treatise on the Diseases of the Eye," in the American edition and in the English edition.

Barth had been directed by the Empress Maria Theresa to teach "two young men as much as he knew and could impart on that subject." Such was the beginning of the noble Vienna school of ophthalmology.

Frick, some time after his return to America, opened an ophthalmic clinic at the Baltimore Dispensary and in 1823 established a course of

lectures in the University of Maryland, which was so systematized as rightly to be considered to have been the first regular course in ophthalmology in America. His book, which he dedicated to Dr. Physick, published in 1823, a more or less formal presentation of those lectures, served as the leading textbook for Americans for several years, and in 1826 an edition was issued in England.

Fig. 11.—George McClellan.

In Philadelphia, in the meantime, through the efforts of certain earnest graduates of the University of Pennsylvania, the Jefferson Medical College of Philadelphia was being organized. Foremost among the founders was George McClellan, a former pupil of Physick, the professor of surgery and anatomy, who because of an interest in ophthalmologic subjects endeavored to devote some time in his general lectures to the exposition of the diseases of the eye.

All those whose names I have mentioned [1] had been under the tutelage of Professor Physick, who properly may be considered to be the "father of American ophthalmology" as well as the "father of American surgery." Therefore, Physick, Dorsey, Gibson and McClellan were the forerunners of ophthalmology in Philadelphia. These four

Diseases of the Eye. 277

CHAPTER X.

DISEASES OF THE EYE.

THE eye, from its delicate and complex structure, and the number and diversity of its diseases, was formerly much neglected, especially in Europe, by the regular members of the profession, and attended to almost exclusively by ignorant and itinerant oculists. Within the last twenty years, however, the value of this important branch of surgery has been duly estimated, in proof of which it need only be mentioned that Ware, Saunders, Gibson of Manchester, Adams, Wardrop, Travers, Vetch, in Britain, and Scarpa, Beer, Schmidt, and others on the continent, have contributed largely by their writings and operations to elevate this department to a most respectable rank. Many of these gentlemen, indeed, forsaking the general practice of their profession, have devoted their whole attention to ophthalmic surgery, and with a result truly honourable to themselves, and glorious to their country.

The most common affection, perhaps, of the eye, is ophthalmia. Of this, therefore, it will be proper first to treat. Ophthalmia is employed by most modern writers as a generic phrase —denoting ocular inflammation. For the sake of precision and accurate discrimination, other terms have been invented, some of them simple and expressive enough, others formidable in the extreme, or altogether monstrous.* To elucidate the varieties

* Nothing short of affectation or pedantry will enable us to tolerate, in many instances, the phraseology of Beer and Schmidt—such as opthalmo-blennorrhœa, blepharo-opthalmo-blennorrhœa, dacryoadenitis, blepharophalmitis idiopathica,

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Fig. 12.—A page from McClellan's "Principles and Practice of Surgery."

surgeons were more or less familiar with medical affairs in London and were much influenced thereby. The great "Moorfields" ophthalmic hospital, established in 1805, had begun to attract the attention of

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1. None was a Fellow of the College of Physicians of Philadelphia, because at that period the fellowship was confined to "physicians."

Americans. An infirmary for patients with diseases of the eye and ear had been opened in New York in 1820, and a similar infirmary was opened in Boston in 1824. In 1821 McClellan opened a "Dispensary for Diseases of the Eye" near Sixth and Walnut Streets. That dispensary struggled along for a year or so, after which it was displaced by "The Pennsylvania Infirmary for Diseases of the Eye and

ISAAC HAYS. M. D.

Isaac Hays

SURGEON TO WILLS HOSPITAL

Editor of the American Journal of the Medical Sciences &c.

Phil.ᵃ Pub.ᵈ by Harrison, 39 So.ʳ 6.ᵗʰ St.

Fig. 13.—Isaac Hays.

Ear," organized in February 1822 at a house on Seventh Street, near Market Street by a group of public-spirited citizens led by Drs. Isaac Hays and George B. Wood. The efforts of these forward-looking men were not appreciated, yet this infirmary continued till about 1829.

During the first thirty years of the nineteenth century the state of medical opinion in America was most unstable. It was a period of reconstruction; practitioners hardly knew where they stood, yet the best minds dared to hope that a scientific era might succeed that marked by the skeptical and uncertain attitude existing among both practitioners and society. In Philadelphia strife and emulation were rife, even among the most cultured practitioners of the day. The forces of

GEORGE B. WOOD, M.D.

Professor of Materia Medica and Pharmacy

IN THE UNIVERSITY OF PENNSYLVANIA

Fig. 14.—George B. Wood.

the University of Pennsylvania were indignant because of the establishment of the Jefferson Medical College. The contemptuous and distrustful demeanor exhibited by the graduates of both these schools

for the practices of those that were self-taught though already well established in the city conspired to disturb the peace of the medical profession, and because of this state of affairs society held aloof from any new projects.

Yet, in spite of these unpleasant conditions, by 1830 there had permeated into the minds of certain thoughtful persons the realization that diseases of the eye should be counted as holding an important sociologic as well as medical significance and that America should follow in the direction already set by London and Vienna and establish institutions for the care of patients so afflicted, as well as to provide opportunities for the clinical instruction of medical practitioners.
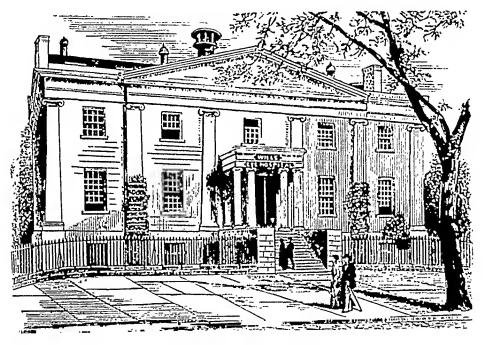
Fig. 15.—The original Wills Hospital.

The Philadelphia pupils of the teachers already named hopefully bided their time while doing what they could to carry out the principles that had been taught them. When, in 1825, a modest grocer named James Wills died, bequeathing about $115,000 of his fortune to charity, part of which was to be spent, in establishing a hospital or asylum to be denominated the "Wills Hospital for the Relief of the Indigent Blind and Lame," the new era they so longed for seemed to have dawned.

After some years of dispute and litigation among the heirs of James Wills, in 1831 the Supreme Court of Pennsylvania decided that the bequest should stand. With the funds available, ground was purchased and a building erected. When the old building on Race Street opposite Logan Square was finished and opened on March 3, 1834, there was left for use and maintenance only $3,000 a year! The first

staff consisted of Isaac Parrish, a youthful but promising recent graduate, the son of a noted philanthropist, who served with ardent devotion until his death in 1852; Squier Littell, a busy practitioner, whose service extended from 1834 to 1863; Isaac Hays, a brilliant practitioner and litterateur, subsequently the editor of the famous *American Journal of the Medical Sciences*, who served from 1834 to 1849, and George Fox, who served from 1834 to 1849. Fox was a good practitioner, but he had many private interests and was a man of affairs as well.

Parrish gave a course of lectures to the students who already began to visit the hospital; these lectures, with Frick's, were the first systematic ophthalmologic lectures delivered in America.

Fig. 16.—Squier Littell in 1866.

Littell had been a student of Parrish. He served as editor of several journals, and in 1837 published "A Manual of the Diseases of the Eye," an English edition of which was brought out by Hugh Houston in 1838. In 1848 a second American edition was issued which represented the experiences which Littell and his colleagues had gained by their continued service at the Wills Hospital. Besides teaching at the bedside and in the clinic, Littell contributed several papers on ophthalmologic subjects.

By 1840 the infant Philadelphia school of ophthalmology was ready to cast off its swaddling clothes. Regular teaching courses (by clinical observation of the patients) were in progress at the Wills Hospital. The members of the staff reported quarterly in the medical journals

the details of the diseases seen at the hospital. Hays devised instruments for operations for cataract. His literary activities were of the greatest helpfulness to American practitioners, and some years later he noted the occurrence of defective perception of color in association with a condition dependent on cerebral disease. Attention began to be

A

MANUAL

OF THE

DISEASES OF THE EYE.

BY

S. LITTELL, Jr., M. D.

ONE OF THE SURGEONS OF THE WILLS HOSPITAL FOR THE BLIND
AND LAME, FELLOW OF THE COLLEGE OF PHYSICIANS
OF PHILADELPHIA, ETC. ETC.

————————

PUBLISHED BY

JOHN S. LITTELL,

PHILADELPHIA.

. . . .

1837.

Fig. 17.—Title page of the first American edition of Littell's "A Manual of the Diseases of the Eye."

called to problems connected with defects of sight dependent on errors of refraction.

The value and influence of Hays', service to ophthalmology cannot be overestimated. To begin with, he brought into the profession nobility of character and a highly cultured intellect, and, possessing

talent for literary expression, he was able to present through the issues of the journals of which he was editor essays and reports of medical topics and cases, offered by Americans, which he had chosen for their scientific usefulness, thereby attracting the attention of the best ophthalmologists both of this country and of Europe. And he analyzed the

THE .

AMERICAN JOURNAL

OF THE

MEDICAL SCIENCES.

ARTICLE I. *Report of Cases treated in the Wills Hospital for the Blind and Lame during the months of October, November and December, 1839, with Observations.* By ISAAC HAYS, M. D., one of the Surgeons.

AMAUROSIS.—There were two cases of this disease, both convalescent, in the house when I took charge of it, and two were admitted during my term of service. One of the former and one of the latter were discharged cured; the other two remained, but somewhat improved. Three of these cases appear to us of sufficient interest to deserve some notice.

Case I. *Partial Amaurosis—Inability to distinguish certain Colours.*— Mary Bishop, ætat. 20, unmarried, segar maker, admitted February 9, 1839. The early history of this case has already been given* by my colleague, Dr. Fox; it will be sufficient, therefore, to recapitulate here merely its prominent features. The patient stated, that she had suffered, previous to admission into the hospital, two attacks of cerebral disease, one in the spring of 1837 the other in the winter of 1837–38. After recovery from the first attack, objects for a time appeared to her double. The second attack left her entirely blind, in which condition she continued for four months. After this her sight began to return, and at the period of her admission into the hospital she could read large print, as the heading of a newspaper. She was of a short, robust stature, full habit, very dark complexion, black hair and hazel irides, flushed face, colour of her cheeks at times almost of a purplish hue; catamenia suppressed. When she first came under my notice, which was

* See No. of this Journal for Nov. 1839, p. 16.

Fig. 18.—A page from the *American Journal of the Medical Sciences,* showing the report of Hays' case of the color-blind girl from the Wills Hospital.

productions from other countries, which analyses were condensed and published with strict regularity for the service of the readers in this country. It was through this editorship that he became important to the practice of ophthalmology, not only in those earliest years but well on in the later days.

Hays, being an ophthalmologist in practice, was able to present to the general reader a topic of special interest in such a manner as to impart an understanding of its importance in relation to medicine, as well as to identify the subject with the growing science of ophthalmology. It must be borne in mind that in those days there was no journal devoted to ophthalmology; the editors of journals of general medicine, however, early realized the growing importance of the specialty and welcomed contributions from practitioners. So one can find chapters on diseases of the eye in the *American Medical Reporter* and the *Journal of the Medical and Physical Sciences*, published in Philadelphia. Frick's articles on conjunctivitis, cataract and artificial pupil appeared in the *American Medical Reporter* for 1821, 1822 and 1823. These early journals were succeeded by the *American Journal of the Medical Sciences*, edited by Hays. In 1826, 1827 and 1828 appeared articles under captions the like of which are to this day a feature of that famous monthly publication. It can thus be seen how Hays advanced the dissemination of the knowledge of ophthalmology in America. Isaac Hays further benefited the native practice by editing for American readers works by English authors, notably Lawrence's famous book "A Treatise on the Diseases of the Eye."

Medical students were encouraged to select ophthalmologic topics as subjects for their graduation theses. Among these theses might be mentioned that of Isaac Cleaver, who graduated from the University of Pennsylvania in 1805, who wrote on "Cataract," dedicating his thesis to Dr. Physick. Another thesis, "The Eye and Vision," by Elisha de Butts, dedicated to Dr. Caspar Wistar, was published in the same year. A third noteworthy thesis, "On the Nature and Treatment of Cataract," dedicated to Dr. George McClellan, was by that destined to be truly great surgeon S. D. Gross, who graduated from the Jefferson Medical College in 1828. The papers here mentioned represented the knowledge obtainable in the times in which they were written.

An event of outstanding importance to the ophthalmology of the whole world occurred in Philadelphia in 1828, when John McAllister, a spectacle maker, succeeded in grinding a planoconvex cylindric lens, a glass for the correction of astigmatism. This lens was used by a man who, by reason of his inability to see distinctly at the distances commonly readily perceived by his contemporaries, believed himself to be near-sighted. It is commonly asserted that Dr. Hays was the first to prescribe cylindric glasses.

Another incident regarding Dr. Hays is that after he had observed defective perception of color in the young woman to whose case reference has already been made he learned of color blindness of congenital origin. In an issue of the *American Journal of the Medical Sciences*

in 1840 he reviewed and analyzed all the reported cases of color blindness from that reported by Dalton in 1794. Hays' reports inspired Dr. Pliny Earle, of Philadelphia, who was himself color blind, to describe in 1845 his case and the cases of the members of his own family, in which for several generations the anomaly had been known to exist.

An item I should touch on is that of the discovery of the tensor tarsi muscle by Dr. William E. Horner, a Philadelphian, the professor of anatomy at the University of Pennsylvania. In a series of minute dissections of the eyelids Horner came on delicate muscular fibers

WILLIAM E. HORNER. M. D.

IN THE UNIVERSITY OF PENNSYLVANIA

Fig. 19.—William E. Horner.

extending along the course of the lacrimal canal from the crest of the lacrimal bone to the lacrimal puncta, unconnected with the orbicularis muscle. This he designated the "muscle of the internal commissure," and he stated that its function was to maintain the lacrimal punctum in contact with the eyeball. His dissections were the marvels of all anatomists. If one recalls Horner's contributions to the minute anatomy

of the intestines, it is easy to comprehend how it was he who found the tensor tarsi muscle. I have more than once seen this discovery credited to another Horner, who lived at a much later time than the days of Horner of Philadelphia. Horner was born about 1790 and lived until 1853. He was succeeded at the university by Dr. Joseph Leidy, beloved by many of the fellows of the College of Physicians of Philadelphia.

The interest manifested by those who took care of "eye cases" but who scorned to be called "specialists" attracted the attention of physicians who in their general practice had begun to notice a relation between local diseases, such as diseases of the eye, and derangement of the bodily health. There was a medical staff attached to the Wills Hospital to assist the more specialized attendants. Perhaps this wide concern with ophthalmic subjects arose through Hays' publication, as already noted, of the reports presented by the surgeons at the Wills Hospital, as issued from time to time in the *American Journal of Medical Sciences*. Yet every one was aware that much that was empirical still existed in the practice of the day and that the approach to the treatment of obscure disease could therefore of necessity be only tentative. The elders could but advise juniors that because of the general ignorance of the anatomy and physiology of the eye only a judicious and discriminating use of the means already at hand should be resorted to rather than the putting into practice of ideas of treatment suggested by incompletely understood manifestations of the pathologic processes observed in that organ. The staff at the Wills Hospital accordingly pleaded earnestly for the further establishment and maintenance of special departments in hospitals in which all phases of ophthalmic medicine might be studied. The highly equipped ophthalmic institutes raising their heads today are but the offspring of the Wills Hospital and the other ophthalmic hospitals opened in the early years of the last century in the cities of Baltimore, New York and Boston.

In the early days at the Wills Hospital the patients were cared for only by being taken into the institution, where small wards had been arranged for them. It was not until some time later that "persons whose infirmities allowed them to attend on the stated days of the surgeons' visits" were accepted for consultation and treatment as outpatients. As the fame of the hospital and the prominence of the city as a medical center extended, the number of applicants rapidly increased day by day, and the dispensary service soon became firmly organized.

It was the "age of clinical intuition," brilliant in its comprehension of the manifestations of the external diseases of the eye. No disorder not visible to the observer's eye was understood, yet one must admire the struggles of observers during that period. With the means at hand all was done that could be, but practitioners waited expectantly till some magician's wand might wave and cause the effulgence of the aided

scientific study to shine and light the way to "modern ophthalmology." That magic wand was the ophthalmoscope, with which truly a new world was opened. The invention of the ophthalmoscope in 1851 affected all the medical sciences. There is not space here to tell how the use of that simple instrument cleared up the mysteries of "amblyopia," "amaurosis" and other vaguely comprehended diseases, and how with it the differential diagnosis between cataract and glaucoma was ascertained; nor is there space to describe the diseases of the ocular tunics observable with it. During the first half of the nineteenth century the achievements in operative surgery placed the surgeons in the front rank of the medical profession and largely contributed to the maintenance of the position of Philadelphia as the medical center of the country. There is not space to tell of the influence that the growing ophthalmologic science had on neurology and of the part investigators in Philadelphia took in the development of the ophthalmoneurology of the world, and, lastly, what the investigation into the state of the refraction of school children in Philadelphia has done in the world-wide preservation of the sight of children and adolescents, not to speak of the comfort and health afforded by spectacles to all classes of society, obtained through the correction of the anomalies of refraction. All these chapters, and more, were developed, if not conceived, in Philadelphia.

In this new birth of ophthalmic medicine the efforts of Philadelphians in time became known by the whole medical world. Today, one can find in the annals of ophthalmologic history the records of the contributions made to the science by those early ophthalmologists of this city, the fruits of which the universe can enjoy.

SUGGESTIONS FOR PREVENTION OF OCULAR AND AURAL SEQUELAE OF MENINGO-COCCIC MENINGITIS

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Dr. Heath has carefully analyzed a large number of cases of meningococcic meningitis and shown the frequency of the different ocular sequelae. All I can add is a comparison between the lesions of this disease and those of suppurative meningitis of aural or nasal origin.

Taking his findings as a basis, I am going to suggest that physicians attempt to prevent the ocular sequelae of meningococcic meningitis by treating the various lesions causing the ocular disturbance at the site of the embolic infarction in a manner similar to that now employed in the treatment of suppurative meningitis.

In dissecting injected heads,[1] I noticed several anatomic facts to which my attention had never before been directed. All have been of assistance to me in the early diagnosis and treatment, as well as in arriving at an understanding of the sequelae, of any form of meningitis.

1. A (a) sensory cranial nerve has a prolongation of arachnoid which surrounds it for some distance after it has entered the dura, so that the anatomist can immediately recognize its sensory character by this arachnoid protection. A (b) motor cranial nerve has no such arachnoid prolongation, losing its covering immediately as it enters the dura, so that a slight displacement of the brain is likely to cause paralysis of a motor nerve, be it the sixth, third or fourth, while all sensory nerves are spared.

2. It is in the subarachnoid prolongations of the basal cistern surrounding a sensory nerve that accumulations of exudate lodge, and these accumulations are the cause of many symptoms. When firmly organized they are the cause of the sequelae of pyogenic meningitis.

3. When I attempted, by gravity, to fill the subarachnoid system with colored fluid, flowing from the inner ear, after I had removed the

1. Eagleton, W. P.: Localized Bulbar Cisterna (Pontile) Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex, Arch. Surg. **20**:386 (March) 1930.

outer labyrinthine wall, I found the communications between the arachnoid spaces in the peripheral prolongations to be so small that the granules of dye pigment held in suspension could not pass, while the watery fluid solution of the dye would pass.

4. It is the obliteration by adhesions of these fine arachnoid passages that prevents the spread of infections to all parts of the system in pyogenic meningitis from the ear or nose.

These anatomic facts are of importance in diagnosing the location of the infection from the symptoms in cases of suppurative meningitis. Adhesions are also the cause of sequelae in patients who recover.

All these anatomic factors play a great but less prominent part in meningococcic meningitis. For it should be emphasized that the germ of epidemic cerebrospinal meningitis acts differently from any other pus-producing germ. The meningococcus (a) is not of great virulence and (b) "has comparatively little surviving power when brought into contact with mesodermal tissues although it (c) can live for some time when in relation to ectodermal cells." [2] And meningococcic infection is primarily "a meningococcic sepsis with a secondary meningeal localization" (Herrick [3]).

While all other pyogenic meningeal infections—streptococcic, pneumococcic and staphylococcic—form a local focus of infection in the bone of the ear or nose, the blood stream is sterile until the terminal stage, that is, when the hemocerebrospinal fluid barrier between the blood vessels and the arachnoid mesh breaks down and allows germs to enter the blood stream. From this time on the patient has not meningitis alone but meningo-encephalitis, from which, in my experience, none recover.

However, in meningococcic infections the blood stream is invaded before the arachnoid spaces, while the cranial nerves are not attacked by pressure, as the vascular nature of the infection causes embolic infarcts or toxins (Wertham [4]) to act directly on small vessels of the ependyma, on the cerebral tissue or on its nerves after leaving the brain.

Thus the ocular motor paralysis resulting from meningococcic meningitis, is due not to pressure from an exudate but to changes in the cerebral tissue itself or damage to its nuclei.

The meningococcus stands in its virulence and mode of invasion midway between the pyogenic germs, such as the streptococcus and the

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2. Greenfield, J. H.: Inflammatory Cells in the Central Nervous System, in Penfield, Wilder: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1229.

3. Herrick, W. W.: Early Diagnosis and Intravenous Serum Treatment of Epidemic Cerebrospinal Meningitis, J. A. M. A. **71**:612 (Aug. 24) 1918.

4. Wertham, Frederic: The Cerebral Lesions in Purulent Meningitis, Arch. Neurol. & Psychiat. **26**:549 (Sept.) 1931.

pneumococcus, and the neutropic viruses, which cause poliomyelitis and encephalitis. However, there is a specific meningococcus serum that acts immediately if it can be brought into contact with the micro-organism.

The meningococcus generally disappears from the blood at an early date.

In the vast majority of the cases observed at the Essex County Hospital for Contagious Diseases, at Belleville, N. J., (a) the blood stream has become sterile before the diagnosis of meningococcic meningitis has been made by examination of cerebrospinal fluid removed by lumbar puncture, although in a few cases (b) the meningococcus was still to be isolated from the nasal secretions.

OCULAR AND AUDITORY SYMPTOMS

The Perineuritis of Meningococcic Infection and the Papilledema of Other Pyogenic Meningitides.—The optic nerve is involved as a perineurositis.[5] There is no papilledema from pressure, in contradistinction to the papilledema in meningitis of pyogenic origin, when the optic prolongation of the third ventricle presses on the chiasm.

The Difference in the Auditory Symptoms.—The impairment of hearing associated with pyogenic meningitis is due to effusions in the arachnoid space; the deafness is never complete, because the eighth nerve withstands pressure well. On the other hand, the deafness associated with meningococcic meningitis is generally complete, because the disease involves the vessels of the nerve tissue itself, either in the labyrinth or in the brain.

With the protective exudate of the posterior fossa which develops in association with labyrinthitis or fracture of the skull, the time of the vestibular reaction to turning is reduced to about 50 per cent, and this continues indefinitely, as I[6] pointed out in 1922, although there are no vestibular symptoms of nystagmus at the time of invasion. However, with the embolic process of meningococcic infection, nystagmus is present for some time immediately after the invasion of the labyrinth.

TREATMENT

Suggestion of Injection of Meningococcus Serum Directly Into the Site of the Lesion.—I suggest that the therapeutist from the beginning of meningococcic meningitis try to differentiate (1) embolic lesions of the blood stream (such as the intra-ocular effusions) from (2) those

5. Lewis, P. M.: Ocular Neuropathies and Amauroses in Meningococcic Meningitis, South. M. J. **24**:101 (Feb.) 1931.

6. Eagleton, W. P.: The Reduction from Turning in Diffuse Lesions of the Cerebrospinal System Pathways Over the Cerebellar Cortex as in Protective Meningitis, Tr. Am. Laryng., Rhin. & Otol. Soc. **28**:231, 1922.

due to cerebral meningo-encephalitis, and that serum be applied as near the site of the lesion as is possible. This utilizes the principle of directly attacking the site of the lesion, as is now the universal practice in the surgical treatment of streptococcic and pneumococcic meningitis from the ear or nose.

In cases in which there is vertical nystagmus, putting serum into the basal cistern should be tried, as in all such cases up to the present time the condition has terminated fatally.

Meningococcic Panophthalmitis.—If emboli lodge in the eyeball the injection of serum into the anterior chamber or even into the vitreous might be tried, as in all such cases the condition ends in blindness and is known to be of blood vessel origin. This procedure was advocated by Netter [7] in 1915.

Intracranial Pressure.—Increased intracranial pressure due to meningococcus meningitis can be relieved surgically. With this treatment I have had a limited, but successful, experience.

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7. Netter, A.: Guérison de l'iridochoroïdite suppurée à méningocoques par les injections de sérum antiméningococcique dans le corps vitré, Compt. rend. Soc. de biol. **78**:90, 1915.

ADENOCARCINOMA OF A MEIBOMIAN GLAND

REPORT OF ADDITIONAL CASES

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The basic architectural feature of a tumor (case 1) described in a previous article in the ARCHIVES [1] seemed to be the band of sebaceous mother cells and sebaceous cells (figs. 4 and 5 of that article). Besides that, cells closely related to pavement epithelial cells were present, which grew between the sebaceous cells (fig. 7 of the article mentioned). In some areas the bands were very broad, and islands of sebaceous cells appeared without definite sebaceous mother cells (seen in the right portion of figure 6 of the aforementioned article), side by side with large areas of cells of the second type (seen in the left portion of figure 6 of the article already referred to). The metastatic tumors consisted of a mixture of the two constituent elements of the original tumor (these are seen in figure 8 of the aforementioned article, which especially demonstrates the sebaceous tumor cells).

The supposed basic architecture of this tumor, the band of cells, was universally present in a second case of a tumor of this kind that I was able to study (fig. 1). The report of this case is as follows:

CASE 2.—An elderly man was treated for some time for a tumor of the superior eyelid; finally, as suspicion arose that the swelling of the lid might be caused by a malignant growth, the eyelid was removed and sent to me for histologic examination. A section from the surface of this tumor showed a massive medullary mass divided into irregular fields by slender septums. Microscopically, the tumor was divided into a number of irregular fields by a skeleton of fine and coarse septums of connective tissue. On these septums—and around the blood vessels—epithelial cells were arranged, generally with their long axis perpendicular to the septums. The nuclei were round or oblong, and occasionally more elongated or irregular ones were seen. They possessed a nuclear wall that colored well and had a delicate structure, with generally one, sometimes two, and, rarely, three or no nucleoli. There was a well developed protoplasm, often already vacuolated, as in higher layers. On top of this more or less regular layer there were further layers of irregularly arranged cells. Often, however, it was hard to distinguish between sebaceous mother cells and sebaceous cells. The nuclei of the latter cells, which generally had also one or two nucleoli, lay further apart in the higher layers, as the cells were larger, the protoplasm being highly vacuolated in the ordinary

Fig. 1 (case 2).—Photomicrograph showing the structure of the tumor.
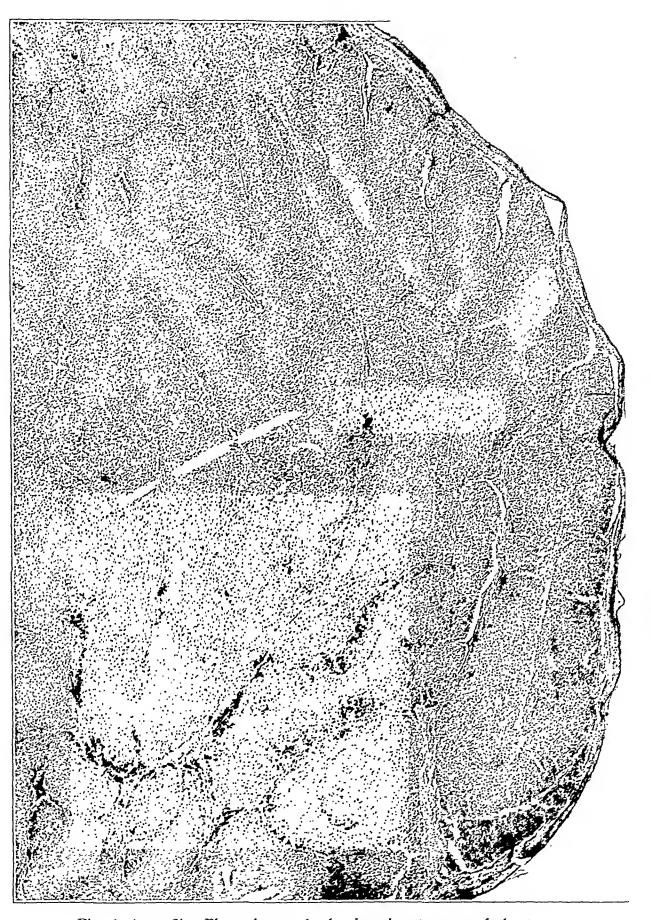
sections by the solution of the alcohol-soluble substances they contained. These were preserved in the frozen sections and stained with sudan III. Double-breaking substances, however, were practically absent. These bands of cells covered the septums of connective tissue, so that in the center a hole might be present, filled with fatty material. Though larger cysts were seen histologically, they were so completely filled with fatty material that macroscopically the tumor seemed to be massive on section. At other places an area marked off by thick walls of connective tissue seemed to be filled by these bands of cells arranged on a slender basal membrane of connective tissue, closely folded together. Most of the nuclei were nearly round or oblong; some, however, were more elongated, and lay in streams. The majority of these cells stained with sudan III as well. The elongated nucleus is atypical for the sebaceous cell, but in this tumor, even though the nucleus was darker and the protoplasm not vacuolated, keratohyaline and horn cells could never be seen, nor could I observe areas of prickle cells, as in the first case. As these nuclei had degenerated, for instance, in the central parts of the cysts, they had become smaller and stained more intensively. Occasionally, cells with a very large nucleus and peculiar cell forms were met with. There were numerous mitosis, more than in the first case. The tumor cells contained glycogen. This was also abundantly present in the surface epithelium, especially in those areas in which
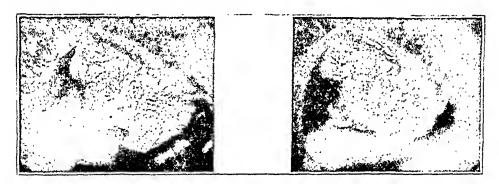
Fig. 2 (case 4).—Stereophotograph of the tumor.

the latter was decidedly hypertrophic over the tumor. The glycogen content of these cells seemed abnormally high compared with that of the cells of similarly colored sections of normal skin.

Gans,[2] in his book on the histologic features of diseases of the skin, gave a diagram (fig. 162) showing "secondary prickle cell proliferation in a case of basal cell carcinoma" and the coloring of glycogen in cases of basal cell carcinoma. The significance of the proliferation of prickle cells and of the high glycogen content of these cells is not yet clear. This high glycogen content of pathologic cells is also present in Paget's disease and Bowen's disease (called precarcinoma by some authors) and in cases of experimental tar carcinoma. If one intends to demonstrate glycogen the material should be fixed in a solution of dextrose and formaldehyde (Romeis [3]).

2. Gans, O.: Histologie der Hautkrankheiten, Berlin, Julius Springer, 1928, vol. 2, p. 327.

3. Romeis, B.: Taschenbuch der mikroskopischen Technik, ed. 13, Berlin, R. Oldenbourg, 1932.
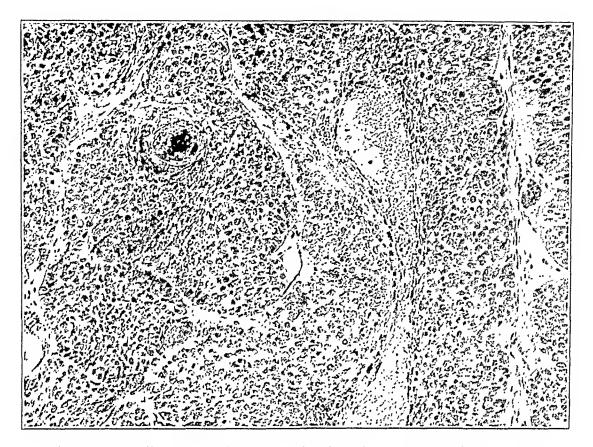
Fig. 3 (case 4).—Photomicrograph showing the structure of the tumor.

Fig. 4.—Histologic aspect of basal cell carcinoma resembling adenocarcinoma.

The observations in the case of this tumor of a meibomian gland induced me to examine the collection of pathologic specimens in the University Eye Laboratory. One tumor was present which was an adenocarcinoma (case 3).
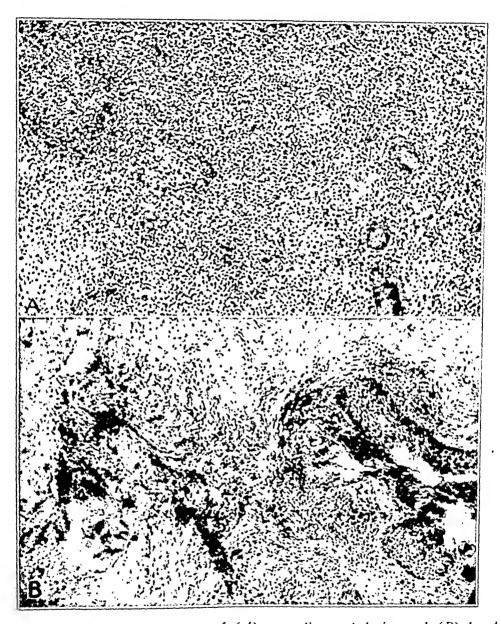
Fig. 5.—Histologic appearance of (*A*) an ordinary chalazion and (*B*) basal cell carcinoma.

CASE 3.—A woman 50 years of age discovered a small nodule in the upper eyelid of one eye three years before she came under treatment. Since then the tumor had gradually increased in size. There were at the first examination a small tumor the size of a pea in the center of the eyelid and some smaller nodules at the temporal side. The eyelid was temporarily fixed on the forehead, and radium was applied. After treatment the eyelid was restored to its normal position.

After seven months a chalazion developed, which was treated in the usual way. Two months later there was a recurrence of the tumor, and the temporal part of the eyelid was removed.

Histologically, the tumor was a typical adenocarcinoma. Many cysts were present, which were filled with granulation tissue like that observed in a chalazion, with many dilated vessels. This is the reaction on necrosis of fat (lipogranulomatosis[4]) and was perhaps caused by the irradiation.

It should be stated here that Lebensohn's[5] patient was cured by radium treatment, so that this treatment, which gives excellent results in cases of other cancers of the lid, should be tried in cases of adenocarcinoma in which it is suitable.

Recently I saw a variety of adenocarcinoma resembling granuloma or papilloma (case 4).

CASE 4.—A few months before the patient was seen the ophthalmologist removed a chalazion. A tumor gradually developed. However, the patient did not return until it had grown to the size shown in figure 2. It was of a much firmer consistency than granulomas, which may develop in cases of chalazion.

A wedge-shaped part of the eyelid was removed, together with the tumor. On section the intrapalpebral part appeared to be sharply marked off against the normal tarsal tissue. Histologically, the tumor was a typical adenocarcinoma of a meibomian gland. The structure was not so regular as that of the tumor in case 2 (fig. 1), and in several places a layer of sebaceous mother cells was not clearly present. Figure 3 shows a body resembling a horn pearl lying among the sebaceous cells.

The tumor did not show infiltrative growth; it was sharply marked off from the surrounding tissue and surrounded by mononuclear cells (lymphocytes and plasma cells). The conjunctiva was decidedly hypertrophic in the neighborhood of the tumor.

COMMENT

In some other carcinomas of the eyelid the number of cells containing substances coloring with sudan III was considerable, and certain areas even resembled adenocarcinoma. Figure 4 demonstrates the histologic aspect of such a tumor, which should be labeled simply basal cell carcinoma. Evidently in this region basal cell carcinoma may show features of adenocarcinoma, and of course there will be found transitional stages in which classification is difficult.

Adenocarcinoma of a meibomian gland is not so rare as is generally supposed.[6] Since my previous paper appeared ten new cases have been described, and I have heard of three cases by personal communication. The majority of these tumors have been taken for a chalazion at their

4. Hagedoorn, A.: Chalazion-Lipogranulomatosis, Am. J. Ophth. **18**:424, 1935.

5. Lebensohn, J.: Primary Carcinoma of Meibomian Gland, Am. J. Ophth. **18**:552, 1935.

6. von Hippel, in Axenfeld, T.: Lehrbuch und Atlas der Augenheilkunde, Jena, Gustav Fischer, 1935.

onset. Therefore it seems wise—if a chalazion has a somewhat unusual consistency—to examine microscopically the material obtained. Thus, in a case of basal cell carcinoma the growth could be detected at an early stage (fig. 5). One must be careful, in examining such material, not to mistake fragments of diseased meibomian gland for carcinoma cells. A few days later the suspected area was removed. The tumor was a basal cell carcinoma, which had apparently originated from the hair follicles. Up to now there has been no recurrence.

The observations in this case show that a tumor suggesting a chalazion is not identical with adenocarcinoma of a meibomian gland. Recently, Vele[7] described a fibrosarcoma of the eyelid which had been considered a chalazion and treated as such.

SUMMARY AND CONCLUSIONS

Adenomas of sebaceous and sweat glands when described by pathologists and dermatologists are classified as nevi in the sense of Darier and Gans. In cases of adenocarcinoma of a meibomian gland there is no doubt as to the blastomatous nature of the growth (though of course it is not impossible that in some cases the tumor may originate from a nevus). As is clear from the foregoing reports and comments, this tumor is not a basal cell carcinoma or a prickle cell carcinoma. It is a special variety of glandular carcinoma and deserves an independent place among other special carcinomas, a place which it has not acquired up till now, as it is not mentioned in the textbooks of pathology. It consists chiefly of bands of sebaceous mother cells and sebaceous cells, or—if it is less regular—of solid masses of sebaceous cells, cysts and papiloma-like growths. Besides that, in my cases cells were present with a tendency to metamorphose as cells of stratified epithelium, which appears to be a second minor characteristic of these tumors. The anlage from which a meibomian gland develops possesses both potencies of differentiation, as the ducts of the glands are lined with cells which assume the characteristics of stratified epithelium near the orifices; even the latent potency to develop a hair may become manifest (distichiasis congenita vera).

7. Vele, Maria: Sui tumori delle palpebre, Boll. d'ocul. **12**:788, 1933.

USE OF SUCROSE PREPARATORY TO SURGICAL TREATMENT OF GLAUCOMA

A PRELIMINARY REPORT

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The preparation of patients who require surgical treatment for glaucoma is as important as that of patients with any other condition which requires operation in which an inclosed cavity of the body is unable to dispose properly of an excess of the products which normally occupy it.

Methods are employed to relieve the distention of the bowel in cases of obstruction, or of the urinary bladder in cases of prostatic hypertrophy, and to reduce the intracranial pressure in cases of disease or trauma that is attended by an increase of contents before surgical treatment is undertaken. An attempt is made to decompress the engorged viscus slowly so that no damage may be done to the vascular channels supplying it. These channels have been functioning under a greater level of pressure than a normal state requires. A sudden release of pressure in a cavity would deprive the walls of the vessels of their accustomed exterior support and thus expose them to possible danger from the increased pressure within themselves until a safe level of pressure could be reestablished. The liability to accident is greater in patients who have vascular changes due to disease or senility, and many patients who have glaucoma are so affected.

Various procedures have been used to decompress the eye slowly and gradually, previous to surgical intervention, in cases of all types of glaucoma in which there is a relatively high tension. The principal objectives are to minimize the danger of intra-ocular hemorrhage from vascular accidents, and to avoid distortion of, or injury to, other intra-ocular structures, which might occur with the sudden release of aqueous at the time of operation. No surgical procedure that we know of can release the pressure slowly enough to accomplish the desired result, because the high level of pressure in the intra-ocular vascular system persists for a considerable time after any type of surgical decompression.

Several procedures have been devised and generally accepted as safe in surgical intervention for glaucoma; each has objectionable features. Paracentesis has a temporary effect only; if surgical decompression is not performed immediately, there is danger of a secondary rise of the tension to the former level or a higher level. The engorgement of the episcleral veins at the limbus may complicate the operation and convalescence by hemorrhage and subsequent hyphemia. The effect of a retrobulbar injection of procaine hydrochloride and epinephrine hydrochloride is also temporary; the fall in the tension is not great or dependable. Any manifest reduction in the tension may be due to the loss of tone in the extra-ocular muscles or to the lessened amount of blood flowing into the eye rather than to any actual loss of excess fluid in the eyeball. The effect of miotics is slow and uncertain, owing, probably, to the congestion of the tissues, which limits their absorption. Purgation and diaphoresis are adjuncts to any of the aforementioned measures and in a debilitated patient might seriously disturb the water balance.

Hypertonic solutions given by intravenous injection have been tried in the effort to accomplish lowering of the tension. In the past the results have been inconstant and unsatisfactory. owing, possibly, to the variable capacity for assimilation, absorption and excretion of different persons. The rationale of such therapy has a plausible hypothetic basis in the concepts concerning the formation of aqueous by either dialysis or filtration.[1] The failures of such therapy have discredited not the theory but rather the agents employed. Hypertonic saline solutions have not been satisfactory because of their rapid excretion by the kidneys and skin and in the expired air and their ready diffusibility out of the vascular channels into the aqueous. Dextrose has been employed with good results in some cases, but the results have been inconstant and therefore undependable. Dextrose is rapidly excreted by the kidneys, and a considerable portion of it is taken up and stored in the liver and muscles in the form of glycogen. Also, it is diffusible into the aqueous.

The ideal preparation must have the properties of not being diffusible into the aqueous, of producing a hypertonic effect in the blood stream for the purpose of directing the osmotic flow away from the aqueous into the blood stream, and then of being excreted completely by the kidneys.

Sucrose possesses many desirable features when used intravenously. The molecule is relatively large and will not readily pass through the capillary wall into the perivascular spaces; it is not broken down and

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1. Duke-Elder, W. S.: Text-Book of Ophthalmology, London. Henry Kimpton, 1932, vol. 1.

therefore remains intact in the blood stream. Sucrose is not absorbed by any of the tissues of the body; it is excreted wholly by the kidneys. It is not contraindicated in diabetes.

There is a similarity in the manner of the formation and circulation of the aqueous and the cerebrospinal fluid. Any preparation which relieves the intracranial pressure in cases of cerebral edema should be in a like manner effective in glaucoma. The intravenous injection of hypertonic solutions of dextrose have been used for a number of years to reduce cerebral edema in cases in which the condition is usually of traumatic origin, but the results have been inconstant and in some instances have been complicated by a secondary rise in the pressure.

The work of Masserman[2] with regard to the use of hypertonic solutions of sucrose in the reduction of the intracranial tension in intact brains led to the employment of this substance by Hahn and his associates[3] for the reduction of excessive pressure in patients with trauma seen at the Indianapolis City Hospital. Having observed the results obtained by Hahn and his co-workers in the use of sucrose in the preparation for operation of patients with injuries of the brain, we were inspired to investigate the possible value of a like preparation in cases of glaucoma in which there was a relatively high intra-ocular tension.

Thus far we have used sucrose intravenously preliminary to thirty operations on twenty patients. Thirty-five or more injections have been made. As a routine, 400 cc. of a 25 per cent solution is injected slowly into the vein, from forty-five to sixty minutes being allowed for the entire quantity to be injected. This substance has been used in cases of all types of glaucoma, whether primary or secondary, in which the tension was over 40 mm. (Schiötz). Thus far our results have been uniformly encouraging. We have found no contraindications to the use of the method, except, perhaps, markedly low renal function. In patients with the latter condition, there was no damage as a result of the use of sucrose, but the effect on the glaucomatous eye was not as marked as in those patients whose renal activity was more nearly normal. There is no contraindication to the use of this agent in diabetes.

The entire range of conditions for which sucrose may be useful has not been investigated as yet, but it is worthy of mention that this substance proved of value in several cases of iridocyclitis complicated by a considerable rise in the tension. The purpose of this report is to present our results with the use of sucrose for reducing the intra-

2. Masserman, J. H.: Effects of Intravenous Administration of Hypertonic Solutions of Sucrose, with Special Reference to Cerebrospinal Fluid Pressure, Bull. Johns Hopkins Hosp. **57**:12 (July) 1935.

3. Hahn, E. V.: Personal communication to the authors.

ocular tension previous to surgical intervention. Five illustrative cases are reported of the twenty which have been studied in the last few months in the ophthalmologic service of the Indianapolis City Hospital.

REPORT OF CASES

CASE 1.—W. D., a Negro aged 67, was admitted to the hospital on Nov. 16, 1936, with noncongestive glaucoma. Vision was limited to counting fingers at 8 inches (20.3 cm.) in the right eye and 6/15 in the left. The visual fields showed a pronounced Rönne step for the right eye and Bjerrum's scotoma for the left. The tension was 45 mm. (Schiötz) in each eye. On November 19 the patient was given 400 cc. of a 25 per cent solution of sucrose intravenously; the entire quantity was administered over a period of about forty-five minutes. At the end of four hours the tension was 15 mm., and after twelve hours it was 12 mm. in each eye. After twenty-four hours the tension had risen to 20 mm., and after forty-eight hours the reading was 20 mm. After seventy-two hours the tension was 28 mm. in the right eye and 32 mm. in the left. On November 23 the tension was 38 mm. in each eye, and an Elliot trephine operation was performed on the right eye. Three hours before surgical intervention, sucrose was given as before, and just previous to the operation the tension was 14 mm. in each eye. There was no complication due to the surgical procedure, but it was noted that the usual "jump" of the upper portion of the iris did not occur when the blade of the trephine entered the anterior chamber. The postoperative course in this eye was uneventful. On November 27 the tension of the eye that had not been operated on had risen again to 38 mm. Miotics were used, without effect. On December 2 an Elliot trephine operation was performed on the left eye. Sucrose was given again, and the tension fell to 14 mm. The operation and convalescence were without note. The tension since that time has remained about 20 mm. The uncorrected vision was unimproved.

CASE 2.—J. G., a Negro aged 70, was admitted to the hospital on Dec. 2, 1936, with glaucoma of the left eye, which had followed extraction of a cataract. Cortical material was incarcerated in the wound. The tension was 48 mm. (Schiötz). Vision was perception of movements of the hand at 2 feet (60.9 cm.). Sucrose was given as in case 1, and the tension fell to 14 mm. Cyclodialysis held the tension within normal limits for six days, after which it returned to a constant level of about 40 mm.; miotics were ineffectual. On December 30 cyclodialysis was again performed. At this time sucrose was not available, and the patient was given 400 cc. of a 25 per cent solution of dextrose. The tension of the eye was not reduced. The second operation failed to bring the tension to normal. After consultation and at the patient's request to be relieved from pain, the eye was enucleated, on Jan. 12, 1937.

CAS 3.—W. S., a Negro aged 53, was admitted to the hospital on Oct. 2, 1936, with glaucoma of the left eye, which was secondary to traumatic subluxation of the crystalline lens. The tension was 85 mm. (Schiötz). There was no perception of light. Sucrose was given as in the other cases, and the tension fell to 22 mm. in four hours, and remained within normal limits for about thirty hours. After seventy-two hours the tension had again reached 85 mm. Sucrose was given again on October 7, and in four hours the tension was 14 mm. Thirty-six hours later, cyclodialysis was performed. The tension immediately before operation was 25 mm. The operation and convalescence were uneventful. The tension has remained within normal limits.

Case 4.—M. Y., a Chinese man aged 63, was admitted to the hospital on Dec. 1, 1936, with a condition that had been diagnosed as acute glaucoma of the left eye. The tension was 53 mm. (Schiötz). Vision was limited to counting fingers at 7 feet (2.1 meters). Miotics did not lower the tension in twelve hours. The patient was given sucrose as in the other cases. In four hours the tension was 14 mm.; the pain had disappeared, and vision had improved to counting fingers at 15 feet (4.5 meters). A basal iridectomy was performed. The tension has remained within normal limits. Vision is now 6/60 in the affected eye.

Case 5.—R. S., a white woman aged 67, was admitted to the hospital on Dec. 1, 1936, with acute glaucoma of the left eye. The tension was 46 mm. (Schiötz). According to the history, a solution of atropine had been instilled into this eye a few days previously. The pupillary diameter was 6 mm. An intensive use of miotics over a period of three days slowly reduced the tension to 18 mm. The tension was still within normal limits at the time of the patient's discharge on December 8. The pupillary diameter was 4 mm. The patient was instructed how to use miotics at home. The medicament prescribed was used faithfully, but when she returned to the hospital on December 13 the tension of the left eye was 52 mm. Sucrose was given, and in two hours the tension was 22 mm. A basal iridectomy was performed, which was followed by an uneventful recovery. The tension has remained normal.

COMMENT

In the cases here reported a variety of antecedent factors were present. The one common observation at the admission of the patient to the hospital was a relatively high intra-ocular tension. The patient in case 1 exhibited a uniform fall in the tension each time that sucrose was used. The reaction of the patient in case 2 made possible a direct comparison of the relative values of a disaccharide (sucrose) and a monosaccharide (dextrose) when employed under almost identical conditions in the same eye. In case 3 it was illustrated how the tension, even when very high, can be brought within normal limits in a short time. The patient in case 4 showed a marked reduction of the tension, with improvement in the congestion, clearing of corneal edema and relief from pain. In case 5 the value of sucrose when the vigorous use of miotics had failed was demonstrated. The reaction to sucrose was prompt and agreeable in all the cases.

The striking feature in the five cases reported in this paper and in others which are not reported here is that the tension was reduced to a relatively constant reading (12 to 22 mm. [Schiötz]) regardless of its height prior to the use of sucrose. We have a working hypotheses for the phenomenon which will necessitate more complete data for its proof. We hope to present this in a future publication.

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HYPERSENSITIVITY TO PONTOCAINE

REPORT OF A CASE

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Pontocaine, formerly called pantocaine (paranormobutylaminoben-zoyldimethylamino-ethanol), in the form of the hydrochloride has gained great popularity as an anesthetic in the fields of medicine and surgery in this country in the last three years. In the practice of ophthalmology it is used in a 0.5 to a 2 per cent solution for instillation into the eye and has replaced many other anesthetics. In Germany it is used in a 1 : 1,000 solution for infiltration anesthesia, and in this country it is widely used in the field of general surgery for spinal anesthesia in a 1 per cent solution. So far in this country no ill effects of the drug have been reported. In Germany hypersensitivity in the eye has been reported by Janke, Mannheimer, Gebb, Rauh and others, and, in the field of surgery, by Schuberth. Since the possibility of hypersensitivity to pontocaine is not generally appreciated, it would seem well to report the following case.

REPORT OF CASE

P. B., a Jewish man of 28, while vacationing observed that vision of his left eye had become hazy and blurred on the nasal side. Since his brother had had a similar experience and was known to have detachment of the retina, he came promptly for examination. He was highly myopic and was found to have detachment of the temporal half of the retina of the left eye, extending to the fovea and without any observable perforations. Nasal hemianopia of the left eye was shown in the field plotted on the perimeter and on the tangent screen. Two drops of a 0.5 per cent solution of pontocaine was instilled into each eye for measurement of the ocular tension with the tonometer. He refused immediate hospitalization for treatment of the detachment. He returned the next day with massive edema of the eyelids, face and parotid regions, diffuse conjunctival injection, and much chemosis of each eye, with edema of the epithelium of the cornea. The swelling, which was accompanied by intense itching at first, had begun several hours after the examination and was so great that the patient was unable to open his eyes.

The patient was then admitted to the hospital, confined to bed and treated with cold compresses and instillations of epinephrine hydrochloride, and a patch test for hypersensitivity to pontocaine was made. Much sloughing of the conjunctiva occurred on the second and third days, and the edema subsided slowly until the twelfth day, when it disappeared. The skin of the arm adjacent to the patch saturated with pontocaine showed erythema after six hours, and innumerable vesicles after twelve hours when the gauze was removed. The next day the epidermis of this area sloughed away entirely. Myriads of eosinophils were noted in smears of material from the conjunctival sacs.

When the edema had subsided it was observed on the tenth day that the retina had become reattached. At the end of the third week the visual fields were plotted by testing with a 1.5 mm. white object and were found to be complete. Three months later the eyes were entirely normal objectively, and the fundi and visual acuity were normal.

Fig. 1.—Photograph of the patient taken on the third day after the instillation of pontocaine.
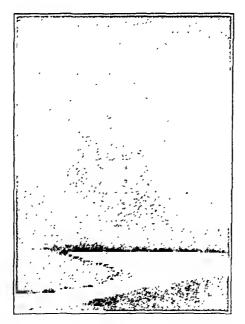
Fig. 2.—Photograph of the inner aspect of the left arm, showing the reaction to a patch test for sensitivity to pontocaine fifteen hours after injection.

COMMENT

Hypersensitivity to pontocaine is disagreeable in the individual case. It must be rare and so should not greatly lessen the position of pontocaine in the armamentarium of physicians. But it should be appreciated that this drug is capable of producing untoward effects, and when

injected subcutaneously or intraspinally in sufficient amounts into a hypersensitive person it may conceivably produce death. Tests for sensitivity to pontocaine should be advocated before it is used for injection in the field of general surgery.

BIBLIOGRAPHY

Gans, H.:   Pontocaine: Eczema Developing After Anesthesia, Hals-, Nasen- u. Ohrenarzt **27**:157-158, 1936.

Gebb, H.:   Pantocaine in Ophthalmology. Ztschr. f. Augenh. **9**:467-469 (Feb.) 1934.

Jahnke, W.:   Pantocaine: Eczema of the Eyelids Following Anesthesia of the Eye, Ztschr. f. Augenh. **83**:112-113 (April) 1934.

Mannheimer, M.:   Hypersusceptibility to Pantocaine Used in Ophthalmic Surgery, Deutsche med. Wchnschr. **59**:85-96 (Jan. 20) 1933.

Marx, E.:   Ophthalmic Reactions to Pantocaine, Klin. Monatsbl. f. Augenh. **89**: 209-214 (Aug.) 1932.

O'Herne, P.:   Complications from the Use of Pantocaine in Ocular Anesthesia, Geneesk. tijdschr. v. Nederl.-Indië **73**:1078-1080 (Aug. 15) 1933.

Rauh, W.:   Experience with Pantocaine, Ztschr. f. Augenh. **82**:134-138 (Dec.) 1933.

Schuberth, O.:   Two Cases of Poisoning from Pantocaine, Zentrabl. f. Chir. **60**: 1822-1824 (Aug. 5) 1933.

Wetmore, F. H.:   Prolonged Toxic Effects of Local Anesthetics—Procaine, Novocaine, and Allied Drugs; Untoward Effects of Nembutal, Canad. M. A. J. **34**:299-300 (March) 1936.

REPAIR OF CHOROIDAL DETACHMENT

REPORT OF A CASE

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Attention has been called by C. S. O'Brien[1] to the great frequency of detachment of the choroid following operation for cataract. This author found it in 93 per cent of his last series of cases. In the article on surgical intervention for glaucoma by Blaess and me[2] there are reported twenty choroidal detachments in one hundred and forty-three eyes operated on for glaucoma.

Choroidal detachment as a rule requires no treatment, for it usually disappears in less than ten days. In the series studied by Blaess and me, two detachments remained, one in a blind eye, for which nothing was done. The other detachment was in a patient with only one eye. The detachment persisted for eight months, when consent was obtained for an operative attempt at repair.

While visiting in Paris in 1932, I saw Professor Magitot do a plastic operation on an eye which had remained too soft after a Lagrange sclerectomy. I cannot recall whether there was a choroidal detachment. Magitot used a piece of Tenon's capsule to cover the scleral defect, but I never learned the result of this procedure.

TECHNIC OF OPERATION

After instilling a 4 per cent solution of cocaine into the conjunctival sac, a 1 per cent solution of procaine hydrochloride was injected subconjunctivally into the upper half of the bulb. An incision was made parallel to the limbus and 8 mm. above it. This incision was about 15 mm. long. The conjunctiva and Tenon's capsule were lifted, and the sclera was exposed to the limbus. The superior rectus muscle was picked up with a squint hook and freely exposed. The central 2 mm. was freed from the remaining portion with a small hook. A single fine silk suture was placed in this portion near the insertion, and it was cut away from the sclera with scissors. Five millimeters of the distal end of the muscle was cut off, and this portion was placed on

1. O'Brien, C. S.: Detachment of the Choroid After Cataract Extraction: Clinical and Experimental Studies, with a Report of Seventy-Five Cases, Arch. Ophth. **14**:527 (Oct.) 1935; Further Observations on Detachment of the Choroid After Cataract Extraction, ibid. **16**:566 (Oct.) 1936.

2. Bothman, L., and Blaess, M. J.: Am. J. Ophth. **19**:1072 (Dec.) 1935.

the sclera across the trephine opening parallel to the limbus. It was fixed to the conjunctiva with three fine silk sutures. The conjunctiva was replaced, and the 15 mm. opening was closed with two silk sutures.

REPORT OF CASE

Mr. E. R. S. was first seen on May 20, 1933, at which time his vision for distance was 1.0—1. He had lacrimation while reading, and he had considerable pain after ten minutes' reading during the previous year. His vision was always better in the morning. There was no history of glaucoma. His left eye was injured forty years before by a piece of straw and was enucleated twenty-seven years before because of irritation and the fear of sympathetic ophthalmia. His right eye had a circumcorneal injection. There were an arcus senilis and a superficial white opacity 2 mm. from the limbus at 8 o'clock. The anterior chamber was shallow; the iris and the pupillary reactions were normal. The peripheral fields as tested with 1 degree white and red targets were essentially normal. The tension (Schiötz) was 30 mm. of mercury, and the pupil was 3 mm. in diameter. Vision of the right eye was 1.0—1 with a —0.50 D. sphere —0.25 D. cylinder, axis 90. On June 10 the tension was 31 mm. There was an Elschnig type 2 excavation of the disk, the excavation reaching the margin between 10 and 11:30 o'clock, and the slope of the wall was suggestive of a beginning glaucomatous excavation.

After 3 drops of eucatropine was instilled the pupil was 4.5 mm. in diameter, and the tension (Schiötz) 35.5 mm. During the period up to October 14 the patient had attacks of smoky vision, and even when the eye was under the influence of pilocarpine the tension rose to 39 mm. The patient was advised to have an Elliott trephine operation, which was done on October 14.

The anterior chamber was not reformed until the fifth day after the operation. On the eleventh day after the operation detachment was seen in the upper nasal and temporal portions of the choroid. On November 22 the choroidal detachment was still present; the best vision of the right eye was 0.4 — 1 with a — 3.00 D. sphere —1.0 D. cylinder, axis 180. The anterior chamber was shallow; the bleb was large, and the tension was within normal limits. On November 28 there was practically no change. On December 20 there were a posterior synechia at 11:30 o'clock and pigment on the anterior capsule of the lens. The incipient cataract, the fields and the choroidal detachment were unchanged. The condition remained about the same, vision becoming gradually less and the lenticular changes slightly more dense, but the tension was only 11.5 mm. on April 30, 1934. On that day the patient was admitted to the hospital, and a posterior sclerotomy was done to withdraw the subchoroidal fluid. There was no change in either the tension or the fields, and vision was 0.2 + 1.

On June 12 the tension was lower, and vision was further reduced. The plastic closure of the trephine opening already described was attempted. The patient had a moderate reaction to the operation, and on July 21 vision was 8/200, but the condition was essentially unchanged. On August 18 the choroidal detachment had disappeared, and the peripheral field for form was entirely normal. The filtrating bleb was much smaller; the anterior chamber was deeper, and the lenticular opacities were slightly increased. Vision was 5/200; the tension was 11.5 mm., and the pupil was 3 mm. in diameter.

The chart shows the field for form as tested with a 2 degree target. The field was taken first on Oct. 26, 1933, twelve days after the Elliott trephine operation; it was taken again on June 9, 1934, three days before the plastic operation, and a third time on August 18, two months after the plastic operation.

The lenticular opacities became more dense, and the fields became smaller. On April 18, 1935, a combined extraction of the lens was done through an incision of the inferior limbus. On Jan. 1, 1936, the secondary cataract was needled. The field was taken a fourth time on Jan. 30, 1937, a 2 degree target being used. On that date vision was 0.8—1.

The cornea was clear; the anterior chamber was deep, and the pupil measured 6 by 6.5 mm. and was keyhole shaped. There was an iridectomy wound at the periphery of the iris opposite 12:30 o'clock. The bleb was cystic and appeared flat. The nasal third of the pupillary space had a dense secondary capsule, but the temporal two thirds were fairly clear. There was a good red reflex. The retina and choroid were in normal position. The disk was pale, and the macula was apparently normal. The tension was 18 mm.
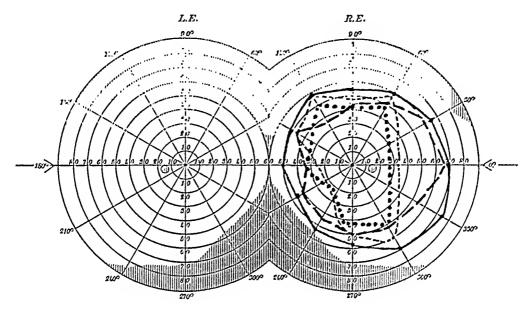
Chart showing the visual field of the right eye on various dates. The line of short dashes indicates the border of the field taken on Oct. 26, 1933, after the trephine operation; the dotted line, the border of the field taken on June 9, 1934, before plastic repair; the unbroken line, the border of the field taken on August 18, after plastic repair, and the line of long dashes, the border of the field taken on Jan. 30; 1937, after extraction of the cataract and needling of the secondary cataract.

SUMMARY

A case of choroidal detachment associated with low tension and constricted fields, which persisted for eight months, is presented. A piece of tendon of the superior rectus muscle was transplanted over the trephine opening, and seven weeks later the choroidal detachment had disappeared and the field was restored to normal.

CONCLUSION

Repair of all choroidal detachments in seeing eyes which persist for three months should be attempted.

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EMBRYOTOXON CORNEAE POSTERIUS AXENFELD

REVIEW OF THE LITERATURE AND REPORT OF A CASE

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In 1920 Axenfeld[1] described a white circular line that he observed on the posterior surface of each cornea, about 1 mm. from the margin, in an otherwise normal person. This he placed in the plane of Descemet's membrane and gave to his finding the name embryotoxon corneae posterius.

There are several earlier reports concerning a similar condition. In 1895 Mager[2] described a ringlike, gray-white opacity 2 mm. in width in the deepest layers of each cornea of a 4½ year old girl.

In 1898 Gloor[3] reported the case of a 6¼ year old girl with many congenital anomalies of the face and extremities. The left eye was normal, but the right cornea showed an opacity 1 mm. in width, near the limbus, extending over two thirds of the cornea. The author did not mention in which layer the opacity appeared, but the drawing showed that it was similar to those described by Axenfeld.

In 1908 Stephenson[4] described the eye of a 5 month old baby with an arcuate light blue strip of tissue which occupied the upper and inner part of the anterior chamber. He stated: "This appears to lie in a plane somewhat anterior to the iris tissue and may even be on the posterior surface of the cornea."

Thier[5] in 1921 added the report of a case of ringlike opacity of Descemet's membrane parallel to the limbus at a distance of one fourth of the radius; otherwise the cornea was clear. The iris was connected with this band.

In 1921 Kayser[6] described two cases of a similar condition. In one the band extended over four fifths of the corneal circumference. In the other case the band was in only the nasal portion. The tissue between the band and the limbus was translucent. No mention was made of iridic bridges.

1. Axenfeld, T.: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **42**:301, 1921.

2. Mager, W.: Wien. klin. Wchnschr. **2**:252, 1895.

3. Gloor: Arch. f. Augenh. **37**:159, 1898.

4. Stephenson, S.: Tr. Ophth. Soc. U. Kingdom **28**:100, 1908.

5. Thier: Arch. f. Augenh. **89**:137, 1921.

6. Kayser, B.: Klin. Monatsbl. f. Augenh. **68**:82, 1922.

In 1927 Remky [7] contributed the first findings on slit lamp examination, in another case in which the opacity was characteristic. He found a narrow, gray-white, slightly wavy band on the back of the cornea, 1 mm. from the limbus, in a 23 year old man with traumatic conjunctivitis. It came out of the angle of the anterior chamber at 6 o'clock and disappeared again into the angle at 11 o'clock. It impressed him as a flat, glassy, opaque line of a rather uniform width, situated on the back of the cornea, giving the impression of a continuation of a line beyond the angle of the chamber.

In 1930 Velhagen Jr. [8] reported under the title "Double Gerontoxon" a complete bilateral ring on the back of the cornea of a 70 year old man with arcus senilis. Not mentioning the hitherto described cases, he considered his finding as a second arcus senilis of unusual position. The added description of the picture seen on slit lamp examination seemed to put this anomaly into the group of those cited.

In 1933 Ida Mann [9] gave an extensive report of three cases of a similar condition.

In the first case the opacity was found in the left eye of a young man with a slight injury to the right eye. The center of the cornea was clear, but a semitransparent membrane extended along most of its circumference on the posterior surface.

In the second case the membrane extended around the lower two thirds of the posterior surface of the periphery of the right cornea. Tissue completely covered the posterior surface of the left cornea.

In the third case the band was adherent to the iris through a segment of the circumference.

In 1935 Rieger [10] described the eyes of a 25 year old girl with ectopic pupils and a slight gray band on the back of the periphery of the cornea, and in 1933 Dudinow [11] found on the back of the cornea of the left eye of a 25 year old man a thick membrane extending from 3 to 11 o'clock.

In 1936 Clapp [12] described the first case of this condition reported in this country, in which both eyes of a 36 year old man were affected. The right cornea was clear in its central portion, but around the periphery was a semitransparent clouding. The left cornea was nearly completely opaque, with general opalescence of the stroma and a translucent membrane on its posterior surface. I am able to contribute an additional case of this type of opacity.

7. Remky: Klin. Monatsbl. f. Augenh. **78**:512, 1927.
8. Velhagen, K., Jr.: Klin. Monatsbl. f. Augenh. **85**:264, 1930.
9. Mann, Ida: Brit. J. Ophth. **17**:449, 1933.
10. Rieger, H.: Arch. f. Ophth. **133**:602, 1935.
11. Dudinow, O. A.: Ztschr. f. Augenh. **81**:336, 1933.
12. Clapp, C. A.: South. M. J. **29**:119, 1936.
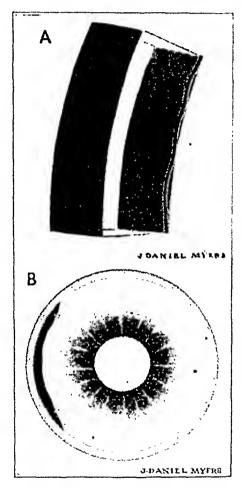
REPORT OF CASE

S. A., a 42 year old white man, came 'to the outpatient department of the Mount Sinai Hospital, complaining of a burning sensation in both eyes. The family history did not reveal any malformations or any hereditary diseases of the eye. The patient had had measles as a child and had frequently suffered from sore throat. For the past six years he had had diabetes. He was married and had two healthy children. The general examination gave negative results. The blood count was normal. Tests of the blood chemistry showed a urea nitrogen

A, slit lamp section through the temporal limbus corneae of the right eye; *B*, front view of the right eye.

content of 8 mg. per hundred cubic centimeters and a sugar content of 210 mg. The blood pressure was 160 systolic and 90 diastolic. Urinalysis showed traces of albumin and a varying amount of sugar. The teeth were normal. The nose and throat were normal. An electrocardiogram suggested hypertrophy of the left ventricle, with probable myocardial involvement.

Ocular Findings.—The palpebral fissures of both eyes were equal. The lids were normal. There was no exophthalmos or enophthalmos. The movements were normal, and there was no nystagmus. The conjunctiva of each eye was slightly injected, but there was no ciliary injection. In the superficial epithelial layers of the right cornea there were three, and in those of the left cornea,

from six to seven, pinhead-sized areas of infiltrates, stainable with fluorescein. The sclerocorneal margin was distinct. There was no arcus senilis. The corneal stroma was clear. On the back of the right cornea, a band 0.75 mm. in width appeared at 8 o'clock, coming from the angle of the anterior chamber and extending in a slightly wavy fashion to 10 o'clock, where it disappeared in the angle of the chamber. It was glassy white and transparent, and in two places there were several fine pigmented spots on each margin. On the back of the left cornea was a similar band, beginning at 7 o'clock and going to 5 o'clock, where it disappeared in the angle, coming out again at 4 o'clock, to disappear at 2 o'clock. The band gave an impression of an empty blood or lymph vessel. The corneal tissue between the band and the limbus was clear. The deep brown iris seemed to be normal. The pupils were round and situated in the center of the iris; they were equal and reacted to light and in accommodation. After instillation of 2 drops of a 4 per cent solution of cocaine hydrochloride they were dilated equally and remained round. The lens was normal. The vitreous was clear. The disk of each eye was normal, but the blood vessels were irregular in caliber, and in the right fundus there were a few scattered small hemorrhages. The tension was normal. The peripheral and central visual fields were normal. Vision was 20/20 in the right eye; it was 20/30 in the left eye, and with correction with a —0.50 diopter sphere equaled 20/20.

COMMENT

The previously described sixteen cases can be divided into three groups: (1) those in which there was a complete ring on the posterior surface of the cornea at some distance from, and parallel to, the limbus; (2) those in which there was a partial band, in which possibly the rest of the complete ring was invisible behind the limbus; (3) those in which there was a broad band of tissue, without free space between the limbus and the band, or a network formation on the back of the cornea.

In all these groups there may be hypoplasia or anomalies of the iris, or more or less fine connections between the iris and the band.

The case reported by me fits into group 2. The name which Axenfeld gave to this finding may be a misnomer. Embryotoxon is generally defined as a congenital opacity of the periphery of the cornea. The normal sharp demarcation between the cornea and the sclera is missing.

Peters [13] in 1909 stated that he considered this type of opacity the most frequent congenital anomaly of the cornea. He observed it histologically to be an overlapping of the cornea by scleral tissue. In the later textbooks it seems to be regarded as identical with arcus juvenilis, in which clear corneal tissue lies between the sclera and the opacity, and hence is but an early acquired gerontoxon, or arcus senilis. In spite of these objections, it seems practical to continue to use Axenfeld's terminology.

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13. Peters, A.: Die angeborenen Fehler und Erkrankungen des Auges, Bonn, F. Cohen, 1909.

As to the etiology, the opinions of the authors are widely divergent.

Velhagen Jr.[8] explained the formation of the ringlike opacity as a colloid chemical phenomenon, the so-called Liesegang's ring.

Clapp [12] considered low grade intra-uterine inflammation as the most satisfactory explanation. In his case the picture was apparently complicated through previous interstitial keratitis, at least in one eye.

Most of the authors agree that the band is a congenital anomaly.

Axenfeld [1] expressed the belief that the opacity is either a ligamentum pectinatum which does not exist normally in man or that it represents incomplete separation of the iris and the cornea.

Ida Mann [9] mentioned the possibility of an increase of the amount and untoward persistence of postendothelial tissue which forms the anlage of the pupillary membrane and the anterior layer of the iris. The origin of this band from mesodermal tissue seems to be certain. The fine connections with the iris and the spots of pigment on each side indicate that this is the case. The similarity to an empty blood or lymph vessel in the case described by me and, probably, in some of the cases formerly reported suggests another explanation.

Schlemm's canal seldom consists of a single lumen; generally there are several in this canal. I suggest, therefore, the possibility that the so-called embryotoxon corneae posterius on the posterior surface of the cornea is nothing else than an atypically located branch of Schlemm's canal. I could not see any blood cells in the one I observed, but the general condition of the patient, who was diabetic, made experiments to produce venous stasis inadvisable.

RETINAL DETACHMENT DUE TO ALLERGY

REPORT OF A CASE

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C. B., a man aged 62, wearing a corrective lens for high myopia and astigmatism (a —6.00 D. sph. —4.00 D. cyl., ax. 70), came to my office in September 1932 with the complaint of blurred vision in the left eye. He had some nodular swellings on his body which he said were caused by eating certain food. Examination of the eyeground showed considerable opacity of the lens and what appeared to be corrugation on the fundus. The blood vessels appeared to be curving and dipping. The patient was told that the fundus had the appearance of beginning retinal detachment. He was put flat on his back and blindfolded for two weeks. As a result the fundus regained its normal appearance, and vision returned to normal.

During the Christmas holidays, three months later, when the patient was eating more than usual, he had nodular swellings on his body and partial loss of vision in his left eye again. Examination of the fundus at this time showed a large detachment in both upper quadrants, which gradually increased until within two days the optic nerve head could not be distinguished. The patient was put on his back and blindfolded for three weeks, but the fundus did not fall back in place as on the previous occasion. He was seen later by a consultant, who felt that the detachment was so great that operation was inadvisable. The patient gave a history of having bumped his head on a door several days previous to this loss of vision, but he felt that it was not the cause of his trouble. This, of course, is questionable.

Four months later, in May 1933, vision of the right eye became blurred, being reduced to 20/100. Five weeks before this vision had been 20/30 with correction, in spite of the opacities of the lens. The fundus was examined, and a corrugated appearance was noted similar to that previously observed in the left eye. The patient was put to bed for two weeks, during which time vision returned to 20/25, and he was able to read Jaeger's test type no. 1.

The patient was seen August 20, four months later; vision of the right eye was found to be 20/25, and he could read Jaeger's test type no. 1 with this eye. Ten days later he returned with the complaint of blurred vision again; swellings on various parts of the body were present simultaneously. Vision of the right eye at this time was reduced to 20/70, with equal impairment for reading. Corrugation again appeared on the fundus, which cleared up with rest in bed and dietary restrictions.

An allergic history of urticaria and nodules on the body, which were at this time associated with the corrugated appearance of the fundus and blurring of the margins of the optic nerve, suggested that his sudden loss of vision might be on an allergic basis.

However, in the following October, because the patient felt that his general condition was good and because the opacities of the lens were increasing in the right eye, the vision of which he was anxious to save, he consented to extraction of cataract. The lens was removed successfully by the intracapsular method.

Vision was 20/30, and he could read Jaeger's test type no. 1 with correction. He progressed well for two months, but in the next three months suffered some impairment of vision, with edema of the optic nerve head and retina. Scopolamine, ethylmorphine hydrochloride and foreign proteins proved of no avail. He was sensitive to atropine.

Vision of the right eye remained about the same (20/100) during the months following, until September 1934. At this time a large retinal detachment developed in the right eye. There was no history of trauma, but the detachment was associated with allergic nodular swellings on various parts of the body.

The patient's history showed the usual diseases of childhood: whooping cough, mumps and measles, two severe attacks of rheumatism, at the ages of 10 and 30 years, and frequent attacks of tonsillitis and infections of the upper respiratory tract. His tonsils were still present. He underwent a prostatectomy in 1930.

He reported having been afflicted several times in his youth with the hives, usually with some accompanying gastric distress. In 1917 and 1918 this allergic disturbance reappeared in the form of nodules and edematous areas, which occurred most often on the back of his neck and behind his ears. Less frequently they appeared on his back, feet, forearms and hands. The patient discovered at this

A and *B*, side and front views of the patient, showing swelling; *C*, appearance of the patient three days later.

time that the ingestion of beans, which he liked and ate frequently, produced marked swellings and gastric discomfort.

At the time of the operation for cataract intradermal allergic tests were made. These showed the following sensitivities: two plus reactions to currant, apple, pear, dill, hops, paprika, black pepper, pimento, sage, vanilla and Fleischmann's yeast; one plus reactions to egg yolk, onion, garlic, banana, ginger, buckwheat, pecan, spinach, beet, horseradish, blackberry, raspberry, strawberry, almond, cherry, lima bean, string bean, peanut, parsnip, parsley, celery, coffee, pumpkin, squash, mushroom, nutmeg, red and green pepper, canary feathers, chicken feathers, goose feathers, duck feathers, cat hair, cattle hair, dog hair, hog hair, horse dander, rabbit hair, sheep wool, orris root, silk and tobacco.

The tests were repeated in June 1934. The following reactions were obtained: four plus reactions to house dust, duck feathers and goose feathers; three plus reactions to orris root; two plus reactions to apple, rye, coffee, chicken feathers, cat hair, horse dander, rabbit hair, sheep wool, kapok, pyrethrum and tobacco; one plus reactions to onion, wheat, lima bean, string bean, cocoa, peach, squash, black pepper, dog hair, cattle hair, goat hair, and cottonseed; plus-minus reactions to grapefruit, potato, peanut and Fleischmann's yeast.

Since that time the patient has been on a diet free from the offending allergens and has had only one serious flareup. This followed a Thanksgiving dinner in 1935, at which time he ate turkey liver. He stated that it tasted unpleasant to

him and caused some gastric distress. The next day he appeared with marked edema of the jaws and cheeks, the greatest reaction he ever had. At the same time blebs developed on the cornea of the left eye, some of which were 3 or 4 mm. in diameter. These gradually disappeared as the swelling of the jaw and lips receded.

The patient had not been tested for sensitivity to turkey liver, but an intradermal test for sensitivity to beef liver was negative. This test has been repeated since, with the same result. The patient had been eating beef liver about two times a week because of an anemic condition, and experiment showed him that some allergic reaction was produced each time he ate it. He was advised to omit liver from his diet and to discontinue the use of cod liver oil in various forms and the halibut liver malt which he had been taking daily as a general tonic. The long-continued absorption of liver preparations may account in some measure for the apparent sensitivity he acquired for liver.

COMMENT AND CONCLUSIONS

The sudden loss of vision and the appearance of corrugations on the fundus associated with nodular swellings on the body, which recurred on numerous occasions before the retina became completely detached, seem to indicate that the pathologic condition in this particular case might have been on an allergic basis.

The appearance of blebs on the cornea coincident with a severe allergic reaction and their simultaneous subsidence seem to indicate that allergic reactions may become localized on or within the eyeball.

Intradermal tests repeatedly failed to show sensitivity to liver, although a reaction in the patient in the form of nodular swellings could be produced by eating it. The patient improved clinically after being on an allergic diet.

The patient has been followed since the fall of 1932, and the case is reported as one in which nodular swellings due to allergic reactions might be present on the cornea and behind the retina and be the specific factor causing retinal detachment.

EFFECT OF CYSTEINE HYDROCHLORIDE ON THE CONJUNCTIVA

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Wherever there are areas of increased proliferation of cells, the concentration of the SH, or sulfhydryl, group is increased. Thus in tumor tissue the concentration of glutathione is high, as shown by Voegtlin and Thompson,[1] Lecloux, Vivario and Firket [2] and Baker.[3] Hammett [4] demonstrated that additions of sulfhydryl substances increased the proliferation of certain roots. According to Hammett and Reimann,[5] proliferation of cells in the skin is accelerated by means of wet dressings containing compounds of sulfhydryl. Lebensohn,[6] who used thiocresol in traumatic corneal ulcers in rabbits, found that epithelialization occurred so rapidly normally that a comparison could not be made.

This report is concerned with the effect of cysteine hydrochloride [6a] on the eye of rabbits and the clinical application suggested from the results. Powdered cysteine hydrochloride was dusted into the conjunctival sac of rabbits daily. This produced reddening of the conjunctiva and superficial abrasion of the cornea, which cleared up in the course of several hours. (The cysteine applied in the form of a paste consisting of equal parts of the powder and water did not cause any denudation of the epithelium.) After three weeks and again after six weeks, parts of the conjunctiva (tarsal and bulbar) and cornea were prepared for microscopic examination.

The tarsal conjunctiva, after three weeks of the application of the cysteine hydrochloride, when examined microscopically showed, instead of the ordinary three or four layers of epithelium, five or six layers. The outer two or three layers consisted of smaller cells with dark nuclei. The sections of the tarsal conjunctiva after six weeks of treatment with the sulfhydryl revealed on examination from nine to twelve layers of cells. The cells appeared larger than the normal cells of the control

1. Voegtlin, C., and Thompson, J. W.: J. Biol. Chem. **70**:801, 1926.
2. Lecloux, J.; Vivario, R., and Firket, J.: Compt. rend. Soc. de biol. **97**: 1823, 1927.
3. Baker, L. E.: J. Exper. Med. **49**:163, 1929.
4. Hammett, F. S.: Protoplasma **7**:20, 1929.
5. Hammett, F. S., and Reimann, S. P.: J. Exper. Med. **50**:445, 1929.
6. Lebensohn, James: Unpublished experiment.
6a. E. R. Squibb & Sons supplied the chemical.
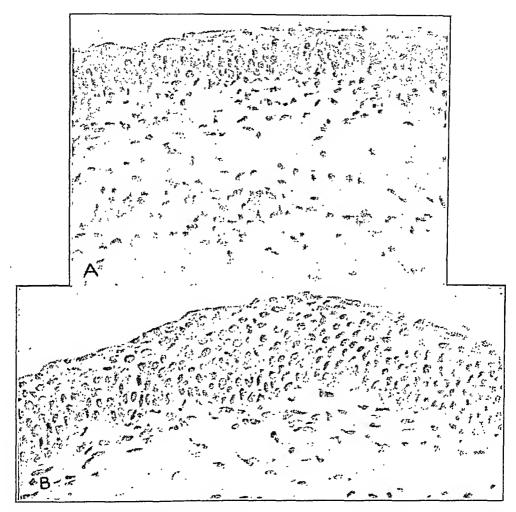
eye, and the nuclei did not stain as deeply as those in the latter. Scattered throughout the epithelium an occasional polymorphonuclear cell was seen, but microscopic examination showed no evidence of inflammation. The epithelium of the cornea was unchanged.

The ability to increase the thickness of the palpebral conjunctiva (at will) may be of value clinically, for it is well known that thick mature mucous membranes (oral mucous membrane and vaginal

A, normal palpebral conjunctiva of the rabbit; *B*, palpebral conjunctiva after treatment with cysteine hydrochloride.

mucous membrane of the adult) are very resistant to the gonococcus. Thus, as was first shown by Lewis [7] and confirmed by others, gonorrheal vaginitis in infants is improved greatly by producing maturation and thickening of the vaginal epithelium by means of theelin. It seems plausible that similar thickening of the palpebral conjunctiva may be of value in treating gonorrheal ophthalmia. In cases in which the condition is unilateral the application of cysteine hydrochloride to the uninvolved conjunctiva for prophylaxis might be considered.

7. Lewis, R. M.: Am. J. Obst. & Gynec. **29**:806, 1935.

HUMAN AUTONOMIC PHARMACOLOGY

IX. EFFECT OF CHOLINERGIC AND ADRENERGIC DRUGS ON THE EYE

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In a comprehensive plan of study of the reactions of .the organs of the human body to mecholyl (acetylbetamethylcholine hydrochloride), prostigmin (the dimethylcarbamic ester of 3-hydroxyphenyltrimethyl-ammonium methylsulfate), benzedrine (benzylmethylcarbinamine or betaphenylisopropylamine) and atropine (the mandelic ester of atropine) a series of experiments were carried out on the eye. The plan of research was that which has been used in this laboratory in studies on sweating, flushing, lacrimation, gastro-intestinal motility, gastric juices, the heart, the pulse rate, the blood pressure and the urinary bladder.[1]

1. Myerson, A., and Ritvo, M.: Benzedrine Sulfate and Its Value in Spasm of the Gastro-Intestinal Tract, J. A. M. A. **107**:24-26 (July 4) 1936. Myerson, A.; Loman, J., and Dameshek, W.: Physiologic Effects of Benzedrine and Its Relationship to Other Drugs Affecting the Autonomic Nervous System, Am. J. M. Sc. **192**:560-574 (Oct.) 1936. Myerson, A.; Rinkel, M., and Dameshek, W.: The Autonomic Pharmacology of the Gastric Juices, New England J. Med. **215**:1005-1013 (Nov. 26) 1936. Myerson, A.; Loman, J., and Dameshek, W.: Physiologic Effects of Acetyl-Beta-Methylcholine (Mecholyl) and Its Relationship to Other Drugs Affecting the Autonomic Nervous System, Am. J. M. Sc. **193**:198-214 (Feb.) 1937. Myerson, A.; Loman, J., and Rinkel, M.: Human Autonomic Pharmacology: VI. General and Local Sweating Produced by Acetyl-Beta-Methylcholine Chloride (Mecholyl), Am. J. M. Sc., to be published. Myerson, A.; Schube, P. G., and Ritvo, M.: The Effect of Acetyl-Beta-Methylcholine (Mecholyl) on the Atonic Colon, Radiology **28**:552-558 (May) 1937. Schube, P. G.; Ritvo, M.; Myerson, A., and Lambert, R.: Human Autonomic Pharmacology: IV. The Effect of Benzedrine Sulfate on the Gall-Bladder, New England J. Med. **216**:694-697 (April 22) 1937. Dameshek, W.; Loman J., and Myerson, A.: Human Autonomic Pharmacology: VII. The Effect on the Normal Cardiovascular System of Certain Sympathomimetic and Parasympathomimetic Drugs—Acetyl-Beta-Methylcholine Chloride, Atropin, Prostigmin, Benzedrine, with Especial Reference to the Electrocardiogram, to be published. Myerson, A.; Rinkel, M.; Loman, J., and Myerson, P.: Human Autonomic Pharmacology: X. The Synergism of Prostigmin and Mecholyl, J. Pharmacol. & Exper. Therap., to be published.

In the working hypothesis (illustrated in fig. 1) we accepted the theory of chemical mediation, which is as follows: (*a*) Acetylcholine is somehow manufactured at the interneuronic synapses of both the sympathetic and the parasympathetic system. (*b*) Acetylcholine is also manufactured at the junction of the second parasympathetic neuron and the reacting cell and brings about whatever activity stimulation of the parasympathetic nervous system produces. (*c*) This chemical activity is carried on in intimate relationship to choline esterase, the mechanism of production of which is unknown, but which seems to be present throughout the tissues of the body. This esterase has the property of entering into chemical union with acetylcholine and thus destroying its effectivity, probably by hydrolysis of the ester radical. The esterases thus bring about an intermittency of activity of acetylcholine. (*d*)
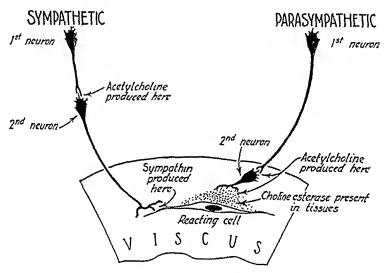
Fig. 1.—Schema of balance between sympathetic and parasympathetic nerves and esterase.

In the sympathetic system, at the junction of the second sympathetic neuron with the reacting cell, a substance akin to epinephrine is produced, called by Cannon sympathin E. This substance produces the effects of stimulation of the sympathetic nervous system.

The working hypothesis may be linked up with the four drugs with which we have experimented as follows (fig. 2):

1. Mecholyl in the main produces the same effects as acetylcholine, although it is much more powerful. The effects of acetylcholine itself on man are difficult to obtain experimentally and with safety.

2. Prostigmin, when given in advance of mecholyl or with it, destroys or inhibits the esterases and, consequently, is a marked synergist of mecholyl.

3. Atropine, when given in advance of mecholyl or during the time of the effects of the latter, abolishes or prevents those effects. Atropine probably does not act on nerve fibers such as the vagus nerve, but

directly changes the chemical reactions which take place between the reacting cell and acetylcholine, so that the effects of acetylcholine are warded off or abolished (Loewi [2]).

4. The other studies of this laboratory have demonstrated further that benzedrine, the effects of which show some divergences from the effects of epinephrine, is mainly adrenergic and that whatever adrenergic effects may take place, such as those of the action of the heart, the blood pressure, the gastro-intestinal motility, and the gastric juices, benzedrine tends to bring them about. Atropine acts as a synergist of benzedrine or of any adrenergic drug by removing the acetylcholine brake or check upon adrenergic activity. Thus, the synergism of benzedrine and atropine differs from that of mecholyl and prostigmin in that the latter synergism is a direct enhancement of effect in the same direction,
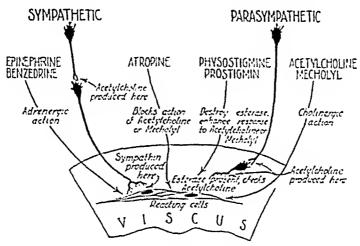
Fig. 2.—Chart illustrating the chemical reactions of mecholyl, prostigmin, atropine and benzedrine.

whereas in the case of the former synergism the parasympathetic brake on adrenergic activities is removed.

HISTORY

The most comprehensive general articles on the autonomic pharmacology of the eye are those by Velhagen.[3] His researches have demonstrated the presence of active chemical substances in the eye, and

2. Loewi, O., and Navratil, E.: Humoral Transmission of Nerve Stimuli: Mechanism of Vagal Action of Physostigmine and Ergotamine, Arch. f. d. ges. Physiol. **214**:689-696, 1926.

3. Velhagen, K., Jr.: Choline Acetylcholine as Physiologic Components of Retinal and Uveal Tissue, Arch. f. Augenh. **105**:573-602, 1932; The Action of the Cholines on the Muscles of the Iris, with Special Reference to Their Double Innervation, ibid. **108**:126-136, 1933; Further Experiments on Active Principles in the Ocular Tissues, ibid. **109**:195-204, 1935.

especially he has shown that there is present both in the aqueous and in the tissues of the iris a substance which in all its reactions corresponds to acetylcholine. This substance is also present in the retina. He stated that acetylcholine and carbaminocholine activate the sphincter of the isolated iris even after atropinization, and the results of these experiments lend support to his theory of double innervation of the sphincter muscle of the iris by sympathetic and parasympathetic fibers, although the dilator muscle is entirely innervated by sympathetic fibers.[4] These drugs act directly on the muscle, which is in line with the results of experiments carried out by many investigators on isolated preparations of muscle elsewhere in the body.[5] The papers reporting their studies should be read in the original because of the scholarly and thorough survey of the literature. Velhagen also pointed out that accumulation of the esterases is a factor in pathologic conditions of the eye, a point which will be considered later as we discuss our own experiments.

Engelhart[6] did not find acetylcholine in the aqueous of the rabbit and cat. He performed, however, a rather critical experiment: If one eye is illuminated and the other kept in the dark, a large amount of the vagus substance, acetylcholine, is found in the illuminated eye, and practically none is found in the dark-adapted eye; a considerable quantity is found in the ciliary body and iris, and this amount is increased by electrical stimulation of the third nerve. Following section and degeneration of the third nerve, the vagus substance vanishes completely from the ciliary body and iris. The importance of Engelhart's experiment lies in the fact that the results showed that light thus acts to produce acetylcholine and that the mechanism is a local one.

Much therapeutic work has been done with acetylcholine in diseases of the eye. Thus, Santonastaso[7] voiced the belief that acetylcholine relaxes the accommodation spasm which reduces distant vision in myopes.

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4. This is not in accord with the results of studies on man.

5. Morison, R. S., and Rosenblueth, A.: The Action of Eserine and Prostigmin on Skeletal Muscle, Science **84**:551-552 (Dec. 18) 1936. Briscoe, G.: The Antagonism Between Curarine and Acetylcholine, J. Physiol. **87**:425-428, 1936. Brown, G. L.; Dale, H. H., and Feldberg, W.: Reaction of the Normal Mammalian Muscle to Acetylcholine and to Eserine, ibid. **87**:394-424, 1936. Rosenblueth, A., and Cannon, W. B.: The Adequacy of the Chemical Theory of Smooth Muscle Excitation, Am. J. Physiol. **116**:414-429, 1936.

6. Engelhart, E.: The Humoral Mechanism of Stimulation of the Third Nerve, Arch. f. d. ges. Physiol. **227**:220-234, 1931.

7. Santonastaso, A.: The Influence of Acetylcholine on Accommodative Spasm, Ann. di ottal. e clin. ocul. **61**:881-889 (Dec.) 1933.

One-tenth gram injected subcutaneously for from six to eight days seems to produce this result, but apparently the most important result is that the near point recedes 1 to 3 cm. It is difficult to account for this result in view of the fact that in hyperopia the near point diminished 24 cm. in one case.

Several other authors have studied the effect of acetylcholine in ametropia and aphakia. The effect of acetylcholine in improving the vision in myopia is believed by Possenti [8] to be due to relaxation of the ciliary muscle, which improvement is in part attributed to the vasodilatation affecting the retinal vessels and hence improving the retinal function.

Acetylcholine has been used in a variety of conditions, mainly arterial in nature—embolism of the retinal artery,[9] blindness due to hemorrhage,[10] quinine amblyopia and amaurosis [11] and homonymous hemianopia of the migraine type [12]—and for its effect on the visual field in diseases of the optic nerve.[13] The intra-ocular pressure, especially in glaucoma, has been studied.[14] Several papers have appeared on the use

8. Possenti, G.: Effect of Acetylcholine in Emmetropia, Ametropia and Aphakia, Ann. di ottal. e clin. ocul. **62**:529-572 (July) 1934.

9. Orr, H. C., and Young, J. H.: Acetylcholine in Embolism of the Retinal Artery, Brit. M. J. **1**:1119-1120 (June 1) 1935.

10. Hartmann, E., and Parfoury, J.: Blindness Due to Hemorrhage Cured by Acetylcholine, Bull. Soc. d'opht. de Paris, February 1934, pp. 56-61.

11. Mazzi, L.: The Action of Acetylcholine in Quinine Amblyopia and Amaurosis, Arch. di ottal. **41**:27-38 (Jan.-Feb.) 1934. Hartmann, E., and Voisin, J.: Quinine Amaurosis Treated with Acetylcholine with Recovery Especially of the Temporal Fields, Bull. Soc. d'opht. de Paris, February 1934, pp. 54-56.

12. Colapinto, G.: Homonymous Hemianopia Cured by Injection of Acetylcholine, Ann. di ottal. e clin. ocul. **59**:883 (Sept.-Oct.) 1931.

13. Cardello, G.: The Effect of Acetylcholine upon the Visual Field in Optic Nerve Diseases, Rassegna ital. d'ottal. **3**:764-793 Sept.-Oct.) 1934.

14. Rycroft, B. W.: The Vascular Control of the Intraocular Pressure: Some Experimental Observations, Tr. Ophth. Soc. U. Kingdom **54**:315-326, 1934. Porhoryles, G.: The Action of Acetylcholine upon the Pressure in the Retinal Vessels, Rassegna ital. d'ottal. **3**:794-805 (Sept.-Oct.) 1934. Villaret, M.; Justin-Besançon, L., and Gallois, J.: The Action of Alpha Methyl-Acetylcholine on the Iris and on Ocular Tension, Bull. et mém. Soc. franç. d'opht. **44**:532-536. Galeazzi, C.: The Action of an Ester of Choline, Carbaminoylcholine, in Normal and Pathologic Eyes, Boll. d'ocul. **13**:1443-1460 (Nov.) 1934. Hartmann, E.: Beneficial Effects of Acetylcholine in Ophthalmologic Practice: Five Cases, Bull. Soc. d'opht. de Paris, June 1933, pp. 414-429. Rossi, V.: Action of Some Derivatives of Choline on the Normal and the Pathologic Eye, Arch. di ottal. **38**:573-585 (Nov.) 1931. Uriarte, A. B.: A New Theory of the Pathogenesis of the Argyll Robertson Sign: Relation to Pupillary Movements, Ann. d'ocul. **172**:672-687 (Aug.) 1935.

of carbaminoylcholine.[15] This drug, according to Velhagen, has the same general effect as physostigmine in lowering the intra-ocular tension and in producing miosis. Acetylcholine has also been used in ophthalmic migraine, with good results, according to Dejean.[16]

MATERIAL

Experiments carried out at the Boston State Hospital, mainly on patients with dementia praecox who were physiologically healthy, and in part on normal persons, showed results which closely correspond to what was expected. The functions of the eye which were studied were: (1) the size of the pupil, (2) the pupillary reaction to light,[17] (3) the pupillary reaction to accommodation, (4) the accommodation of the lens, (5) the intra-ocular tension, (6) the width of the palpebral fissure and (7) the retinal vessels.

1. *Mecholyl.*—The concentration of mecholyl necessary to bring about results on the eye varies from patient to patient remarkably, although this is true of the autonomic pharmacology of man in general. It is perfectly safe to give a 10 to 20 per cent solution. The latter dose will bring about the full effects in any eye, so far as our experiments showed. In some persons a 1 per cent solution will work adequately. It is impossible to predict in advance, so far as our work revealed, what the sensitivity of the individual eye will be. Starting with an average dilatation of from 6 to 7 mm. in diameter, the pupil becomes the size of a pinpoint in twenty minutes, if a sufficient concentration is given.

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15. Wilenkin, M.: Effect of Carbaminoylcholine on Intra-Ocular Pressure and on Width of Pupil, Klin. Monatsbl. f. Augenh. **96**:84-90 (Jan.) 1936. Villaret, M.; Justin-Besançon, L.; Schiff-Wertheimer, and Gallois, J.: The Esters of Choline in Ophthalmology, Arch. d'opht. **49**:129-165 (March) 1932. Velhagen, K.: The Ocular Pharmacology and Toxicology of Carbaminoylcholine, Arch. f. Augenh. **107**:319-344, 1933; The Clinical Use of the New Miotic Carbaminoylcholine in the Treatment of Glaucoma, Klin. Monatsbl. f. Augenh. **92**:472-483 (April) 1934. Miloro, A.: The Effect of Doryl in the Treatment of Glaucoma, Ann. di ottal. e clin. ocul. **63**:780-793 (Oct.) 1935.

16. Dejean, C.: Acetylcholine in Ophthalmic Migraine, Presse méd. **40**:1950-1951 (Dec. 24) 1932.

17. In this paper the classic theory of the light reflex is accepted, namely, that it is mediated by light falling on the retina, and the nervous stimulation thus set up travels until it finally reaches the nucleus of the oculomotor nerve, passing along this pathway to the ciliary ganglion, from which the stimulus is carried to the iris and brings about a contraction. An important paper of Uriarte's (A New Theory of the Pathogenesis of the Argyll Robertson Sign: Relation to Pupillary Movements, Ann. d'ocul. **172**:672-687 [Aug.] 1935), in which he built up the hypothesis of the sympathetic reflex to light, or, rather, to darkness, as more primitive and important, is in line with some work which we have done on the Argyll Robertson pupil. The classic theory does not correspond to the clinical fact, but for the moment we accept it as relevant to our experiments.

The preliminary effect is to dilate the superficial vessels and also the retinal vessels. The eye becomes somewhat watery. These effects disappear in one-half hour. There is no pain. The full effects of mecholyl may be summarized thus: (a) The pupil becomes the size of a pinpoint. (b) The reaction to light is preserved up to the time when the pupil becomes the size of a pinpoint. This is also true of the consensual reaction to light. (c) The accommodation reflex of the pupil is also maintained in a similar way. (d) The intra-ocular tension drops in the normal eye from 3 to 5 mm., according to the tests carried out with the Schiötz tonometer. (e) The fissure becomes definitely narrowed. (f) The retinal vessels, arteries and veins dilate.

Accommodation of the Lens: Invariably with sufficient doses the near point diminished, and the ability to read at that point improved. This was especially marked in the presbyopic patients. In several of the patients who were unable to give an exact account of their reactions, it was obvious that the ability to focus on near objects was definitely improved. Thus, one person was unable to read any of the lines on the Jaeger test type card. After mecholyl was given, he was able to read, although with difficulty, the words on any of the lines on the card. The improvement is not associated with a sense of clearness or ease; that is, although through the action of the ciliary muscle the lens apparently is capable of increasing its convexity, that increase is not spontaneously or easily brought about.

Relationship of Effect of Mecholyl with That of Atropine: Atropine sulfate, when instilled in advance of mecholyl, entirely prevents the action of the latter. This is in harmony with work done on the blood pressure, sweating, the gastro-intestinal motility and the gastric secretions. If atropine sulfate is administered at the height of the reaction to mecholyl, the effects of mecholyl cease and are displaced by the effects of atropine within a few minutes. If atropine sulfate is given in advance of mecholyl, the latter, when instilled in the eye, produces no results the first day. On the second day the effect of mecholyl is contraction of the pupil, but this effect disappears rapidly and is replaced by the effect of atropine. On the third day the effects of mecholyl persist for a longer period, but when these disappear the effects of atropine still are partly present. On the fourth day the effects of mecholyl are normal.

2. *Prostigmin.*—A 1 per cent solution of prostigmin is the minimum dilution which we have found to be effective in producing constriction of the pupils. An optimum concentration is about a 5 per cent solution. A 10 per cent solution does not do any damage to the eye. There is some subjective stickiness of the lids for a short time. The vessels do not dilate to the extent that they do when mecholyl is instilled. The pupillary response is not so prompt, but is complete, and the reactions

resemble those of mecholyl closely. In other words, one would be willing to state on the basis of the theory here postulated that enough acetylcholine is constantly manufactured in the eye so that when the esterases are removed by prostigmin the effects of acetylcholine are produced. The intra-ocular tension diminishes according to the concentration of prostigmin given. A 1 per cent solution causes practically no effect. With a 5 per cent solution a marked effect is obtained; that is, in the normal eye the reduction of the tension is from 3 to 5 mm. The decrease lasts from two to three hours or more, there being a gradual return to the normal. This follows the pupillary change. The effect on accommodation is marked, especially in presbyopic patients. We cite the following case as illustrative of this:

The patient was A. M., a man 55 years of age.

1. Normal Condition of the Eyes Without Prostigmin: The pupils were about 3 mm. in diameter and reacted well. Vision of the right eye was 20/30, and with a + 1.25 D. sph. —37 D. cyl., ax. 15, was 20/20. Tests for accommodation showed that without correction for distant vision the patient could not read even large type, such as Jaeger's test type no. 6 and no. 7, at 12 to 15 inches (30.5 to 38 cm.) with either eye. With correction for distant vision he could read Jaeger's test type no. 6 at 12 inches with each eye. With additional correction for the presbyopia (a + 2.50 D. sph.) he could read Jaeger's test type no. 1 at 6 inches (15 cm.).

2. Condition of the Eyes with Prostigmin: The right pupil was practically the size of a pinpoint. The left pupil was not so small (a smaller amount of prostigmin was instilled into the left eye), measuring about 1 mm. in diameter. There was no reaction in either pupil.

Vision of the right eye was 20/20, and with a + 1.25 D. sph. —50 D. cyl., ax. 90, was increased to 20/15. Vision of the left eye was 20/30, and with a + 1.50 D. sph. —50 D. cyl., ax. 90, was 20/20 +. Without a glass the patient could read Jaeger's test type no. 2 at 10 inches (25 cm.) with the right eye and Jaeger's test type no. 4 at a distance of 12 inches with the left eye. With correction for distant vision he could read Jaeger's test type no. 1 at 10 inches with the right eye and Jaeger's test type no. 2 at 12 inches with the left eye.

It will thus be seen that in the right eye, which was fully prostigminized, the accommodation of the lens for the near point increased from 4 to 5 diopters and that in the left eye, from 3 to 4 D. The effect started with blurring for distant vision, so that for a short time the subject was near-sighted. There was some headache at this time. Later on, the headache disappeared, and for several hours the subject was able to read without glasses. The next morning there was some increase in the capacity to accommodate, but this was not sufficient to allow him to dispense with glasses.

In another presbyopic patient, previously mentioned in the section on mecholyl, who was unable to give a complete account of his reactions, there was marked improvement, as evidenced by the fact that he could read all the lines on the Jaeger test type card.

Synergism of Mecholyl and Prostigmin: This is definite and marked. A subminimal dose of mecholyl (a 1 per cent solution) given to a person in whom this produces no result plus a subminimal dose or

concentration of prostigmin (a 1 per cent solution) produces a marked result, with complete miosis, a definite reduction in the intra-ocular tension and increased ability for accommodation.

3. *Benzedrine Sulfate.*—The instillation of a 0.125 to 1 per cent solution of this drug is necessary to produce characteristic effects. In this instance, as in others, no prediction can be made in advance as to the sensitivity of the subject. The effect is characteristic. There is a slight reduction in the sensitivity of the cornea. There is a moderate injection, which lasts for a few minutes. The pupil dilates to the point of complete mydriasis in from fifteen minutes to one-half hour, the dilatation varying with the amount of the drug given and the concentration. With dilute solutions the reaction of the pupil to light becomes diminished, and this diminution of reaction becomes more evident as the solution of benzedrine sulfate is made more concentrated, so that if a solution of 10 per cent is given the reaction to flash-light practically disappears. However, the reaction to daylight still remains, in counterdistinction to the effect of atropine.

At this point we wish to make note of an extraordinary effect on the Argyll Robertson pupil. When a dilute solution of benzedrine sulfate (a 0.125 to 0.5 per cent solution) is instilled into the eye, the Argyll Robertson pupil enlarges, and the reaction to light reappears; that is to say, such a pupil, which does not react to light, which reacts to accommodation and which is miotic, dilates after the instillation of benzedrine sulfate, enlarges in darkness and contracts with difficulty in reaction to flash-light, but easily, although gradually, in reaction to daylight, so that while it is not normal in the rapidity of its response, it corresponds to the normal in the quality of its response. This subject will be taken up in a separate paper. In a certain sense it confirms Uriarte's conclusions about the Argyll Robertson pupil.

The pupillary reaction to accommodation is likewise impaired after the instillation of benzedrine sulfate, and in this respect the effects of the drug closely resemble those of atropine. In dilute solutions the reaction of the pupil to accommodation is retained.

Accommodation of the Lens: In the younger patient the capacity to focus on the near point was greatly diminished, while distant vision remained without any definite change, so far as our tests showed. Clearness of vision was greatly impaired at all points. In the case of the presbyopic patient A. M., near vision entirely and rapidly disappeared, while distant vision, although not clear, was not greatly changed.

Before any change takes place in the pupil, the palpebral fissure becomes definitely widened. This effect is constant when benzedrine sulfate is given and appears even when very dilute solutions, such as a 0.125 per cent solution, are used. Benzedrine, like epinephrine and cocaine, by its local stimulation of Müller's fibers, widens the fissure and thus

causes the appearance of exophthalmos. This effect is regularly produced and takes place often before the pupil dilates. While one often gets the appearance of exophthalmos, careful studies carried out for us by Dr. Benjamin Sachs with the exophthalmometer showed that there is no true exophthalmos and that its appearance is an illusion created by the enlarged pupil and the widened fissure. Similarly, there is no enophthalmos created by mecholyl, although the fissure narrows and the pupil becomes smaller. The appearance of enophthalmos is striking, but, as has already been mentioned, the measurements carried out by Dr. Sachs failed to show any.

The intra-ocular tension is increased from 2 to 4 mm., according to the concentration.

In Combination with Atropine: The synergistic effect of atropine and benzedrine showed up strikingly in our experiments. A markedly subminimal dose of atropine sulfate (a 1 : 2,000 solution), used together with a 1 : 1,000 solution of benzedrine sulfate, within a few minutes produces marked dilatation of the pupil, which is reactionless to light and accommodation. The accommodation of the lens disappears. The intra-ocular tension is only slightly increased.

4. *Atropine Sulfate.*—The effects of atropine are too well known to need any detailed exposition in this paper. Suffice it to say that in our experiments the instillation of a 1 per cent solution of atropine sulfate produced complete mydriasis and the disappearance of the pupillary reflex to light and accommodation. The accommodation of the lens disappeared, and the intra-ocular tension increased. However, the synergistic effects of this drug with benzedrine are important enough to reemphasize. The complete abolition of the effects of mecholyl by atropine appears as regularly as theory demands. The abolition by the use of atropine of the effects of prostigmin in combination with mecholyl is neither so striking nor so complete. Apparently the addition of prostigmin modifies the action of atropine to a certain extent. This has appeared to be the case in our work on the heart. The abolition of the esterases by prostigmin, if this is its total effect, seems to permit a longer effect of mecholyl.

It is important to note that atropine does not widen the fissure, and in this respect is not synergistic with benzedrine. This illustrates one of the principles of autonomic pharmacology, namely, that atropine has no direct sympathetic stimulation but produces its effects by checking or inhibiting the action of acetylcholine. If a sympathetic influence is present, then, the brake being removed from its activity, an increased sympathetic effect is noted. On the other hand, as in the case of Müller's muscle, as there is no brake to remove, since there is no stimulation of acetylcholine normally present, the activity of Müller's muscle is not changed, and the fissure remains unaltered.

In the first place, the eye is the only organ in which experiments with the drugs used by us can be made without the creation of general effects. Apparently the drugs are absorbed and destroyed by the local tissues and structures without any noticeable absorption into the general circulation. Moreover the eye is capable of withstanding a dose of combined mecholyl and prostigmin which would be fatal if injected under the skin. Thus, by mistake, 2 drops of the 10 per cent solution of prostigmin plus 2 drops of the 10 per cent solution of mecholyl were given intradermally to a patient. Within fifteen seconds he was in collapse, and only the prompt administration of atropine and epinephrine corrected the error. Yet the eye suffers no ill effects from a dose which overwhelms the entire body.

Moreover, when mecholyl is introduced into the general circulation the eye partakes in much lesser measure and after a longer interval than do the other organs. A dose of mecholyl sufficient to lower the blood pressure 40 to 60 mm. of mercury, to change the gastric secretion from $+20$ to -50 HCl, to bathe the whole upper part of the body in a profuse alkaline sweat and to shift remarkably the tonus of the smooth muscles of the rest of the body has almost no discernible effect on the eye. The same is true of benzedrine and epinephrine. The general effects of these drugs, when subcutaneously administered, are enormously spread throughout the organism, but the eye does not partake of the disturbance in anything like proportionate measure. Atropine, although it affects the eye, produces its general effects much earlier than its ocular changes. Since prostigmin is largely silent in its effects, except on the atonic intestine, little can be said concerning the relationship of the general and the ocular effects of this drug.

Concerning the pupillary size, the facts we adduce are in line with the general knowledge. So far as we know, benzedrine has never been used as a mydriatic, nor has prostigmin been used as a miotic. The synergism between benzedrine and atropine seems clinically useful, since it is possible to produce complete cycloplegia of short duration, with little increase in the intra-ocular tension. The method of iontophoresis in respect to mecholyl seems useful in bringing about gentle effects in the eye, as elsewhere in the body.

The intra-ocular tension is, in certain respects, a resultant of the balance between cholinergic stimulation, choline esterase and adrenergic stimulation and closely corresponds to such functions as the heart rate and the secretions of the stomach. The pressure lessens with cholinergic stimulation (after the instillation of mecholyl) or with the inhibition of the esterases (after the instillation of prostigmin). It increases when the cholinergic substances are shut off from action (after the instillation

of atropine sulfate). It increases when there is adrenergic stimulation (after the instillation of benzedrine sulfate). Whatever the hidden modus operandi of this relationship to the autonomic pharmacology may be, the fact that the effect of these drugs on the intra-ocular tension stands in a classic and expected relationship seems to us to be certain. (See Imachi.[18])

The reaction of the accommodation of the lens does not correspond to expectation. Anatomically, the lens is a unilateral mechanism entirely innervated by the parasympathetic fibers from the midbrain, yet benzedrine has an effect on accommodation of the lens. However, an explanation can be found for this effect, namely, the fact that benzedrine acts directly on the ciliary muscle to relax this muscle; that is to say, it does not necessarily follow that the effect of the instillation of substances into the eye actually proves anything about their normal nervous relationships. Thus, the ciliary muscle may be capable of responding to a sympathomimetic drug brought directly into relationship with it, as by instillation, and yet have no normal sympathetic action. While it would be expected that mecholyl, by positive stimulation of the fibers of the ciliary muscle, would bring about an increased curvature of the anterior surface of the lens, thus heightening the power of accommodation, the effect of prostigmin, while expected in certain measure, exceeds expectation in quantitative relationship. In other words, the esterases seem to play an exceptional rôle in the contraction of the ciliary muscle. One might assume that while acetylcholine is being produced in the presbyopic eye, the amount of esterases present prevents accommodation, and that prostigmin, by removing the inhibitor, allows the acetylcholine normally produced to act with vigor on the ciliary muscle and thus on accommodation.

SUMMARY

1. Mecholyl (acetylbetamethylcholine hydrochloride), in a 1 to 10 per cent solution, when instilled into the conjunctival sac constricts the pupil to miosis but permits the reaction of the pupil to light until extreme miosis has been reached. It increases the accommodation of the lens and decreases the intra-ocular tension. It narrows the palpebral fissure and probably dilates the blood vessels of the retina.

2. Prostigmin, in the eye, as elsewhere in the body, is a synergist of the effects of mecholyl, besides having the same general effects when used by itself. It has an especially noteworthy effect on accommodation, increasing the power of the lens to accommodate to the near point.

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18. Imachi, K.: Experimental Investigation of Ocular Pressure and Its Relation to the Tone of the Vegetative System, Acta soc. ophth. jap. **36**:79 (July) 1932.

3. In the eye, as elsewhere in the body, atropine sulfate prevents the action of mecholyl and abolishes its reactions when these are present. Therefore it dilates the pupil and paralyzes the constrictor muscle of the iris, thus bringing about an absence of reaction to light. It increases the intra-ocular tension, since the parasympathetic nervous system seems to lower this tension. In addition it paralyzes the accommodation of the lens to the near point, since the ciliary muscle, which brings this about, is cholinergic in function. It does not widen the palpebral fissure.

4. Benzedrine sulfate acts adrenergically in all respects on the eye. It dilates the pupil and diminishes the light reflex, although it does not abolish the reaction to sharp daylight. It increases the intra-ocular tension and diminishes the capacity of the lens to accommodate to the
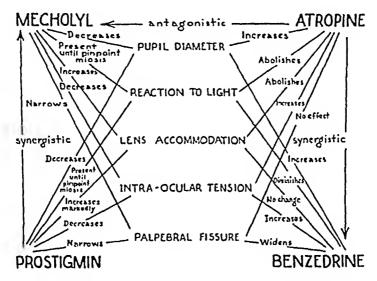
Fig. 3.—Chart showing the effects of mecholyl, prostigmin, atropine and benzedrine on the functions of the eye.

near point. It widens the palpebral fissure strikingly but does not produce exophthalmos. It narrows the arteries and veins of the retina. It reacts synergistically with atropine sulfate. A subminimal dose of benzedrine sulfate combined with a subminimal dose of atropine sulfate will bring about marked miosis and cycloplegia, with little change in the intra-ocular tension.

Merck & Co., Inc., furnished the mecholyl used in these experiments; Smith, Kline & French supplied the benzedrine sulfate, and Hoffmann-La Roche, Inc., the prostigmin.

Dr. Benjamin Sachs checked and controlled the studies by his own observations, especially in respect to the accommodation of the lens under the influence of prostigmin, the question of exophthalmos and enophthalmos, and the intra-ocular pressure.

TUMOR OF THE OPTIC CHIASM AND OPTIC NERVES

REPORT OF A CASE

JESSE M. LEVITT, M.D.

BROOKLYN

Primary tumor of the optic nerve in general is rare. That arising at the intracranial portion of the optic nerve and the optic chiasm is particularly so. This usually occurs in childhood and sometimes is associated with generalized neurofibromatosis, or Recklinghausen's disease. The most significant and often the only signs are ocular. Primary atrophy of the optic nerve associated with divergent strabismus in one eye is a common finding. Occasionally there is a superimposed papilledema. Studies of the visual fields show either total blindness in one eye and hemianopia in the other, or bitemporal or homonymous hemianopia. The roentgen rays are helpful in diagnosis. The sella turcica is often deformed and the optic foramen enlarged. The growth of the tumor is slow; invasion of the orbit producing exophthalmos occurs late. Surgical intervention is not indicated. The diagnosis is often difficult, and surgical intervention is undertaken as an exploratory measure. The usual lesions which give rise to difficulty in diagnosis are meningioma and craniopharyngioma, both amenable to surgical intervention. Roentgen treatment is beneficial.

Lundberg [1] recently wrote an excellent monograph dealing with the clinical and pathologic aspects of primary tumor of the optic nerve and chiasm. The ocular diagnosis in many cases which later were proved to be instances of tumor of the nerve and chiasm was retrobulbar neuritis. The point is stressed that many cases of unexplained blindness or atrophy of the optic nerve may belong to this category, particularly in the early stage, in which field defects and other signs are lacking. The early age incidence, the loss of vision preceding exophthalmos and the relatively small degree of limitation of ocular motility are presented as important points in distinguishing intrinsic tumor of the optic nerve from meningioma.

1. Lundberg, A.: Ueber die primären Tumoren des Sehnerven und der Sehnervenkreuzung, Inaug. Dissert., Stockholm, Nordiska Bokhandeln, 1935.

REPORT OF CASE

History.—Mrs. E. H., 43 years of age, was examined by me at the ophthalmologic outpatient department of the Jewish Hospital of Brooklyn in January 1936 and hospitalized in May. She complained that the vision of the left eye had gradually failed over the course of ten to fifteen years, reaching a state of total blindness five months prior to the first examination. The same eye had become more prominent within the past three months. Drooping of the left upper lid of recent onset was also observed by the patient. There were no general symptoms. The past and the family history were not significant.

Ocular Examination.—Vision of the right eye was 20/50 and with correction was 20/20; vision of the left eye was nil.

There was ptosis of the left eye, the lid covering the upper half of the cornea. The globe was slightly divergent and definitely proptosed. Rotation nasally and downward was markedly limited. The pupil was slightly dilated and did not react to direct light but reacted consensually. There was no pulsation of the globe or bruit. Palpation of the orbit gave negative results. Corneal sensation was unimpaired. The media appeared entirely clear. The optic disk was completely white and clearly outlined; the retinal blood vessels were normal.

The right eye was normal in every respect except that examination of the visual field revealed temporal hemiachromatopsia for red and green.

Fig. 1.—Photograph showing ptosis and exophthalmos of the left eye.

With the Hertel exophthalmometer the reading for the right eye was 13 mm. and that for the left eye 21 mm.

General Study.—Physical examination revealed a fairly well nourished white woman. The findings were limited to the eye. The blood pressure was within normal limits. All the reflexes were normal. No facial weakness and no sensory or motor disturbances were present. No neurofibromatosis was found. Examination of the nose, throat and sinuses, including roentgen study, did not disclose any significant pathologic changes.

Roentgen studies of the skull (figs. 2 and 3) showed a normal cranial vault, a shallow sella turcica, erosion of the anterior and posterior clinoid processes and erosion of the left sphenoid ridge. The left optic foramen appeared to be enlarged to twice the size of the right.

The Wassermann and Kahn tests of the blood gave negative reactions; the blood chemistry and the hematologic picture were normal, and urinalysis yielded negative results.

Course.—Craniotomy was performed, and a massive chiasmal growth was exposed. No attempt at removal was made. The patient died on the twelfth postoperative day.

Postmortem Examination.—This showed an unusual picture. The left optic nerve was about the size of a lead pencil, discolored, cystic and neoplastic in appearance. The optic chiasm was replaced by a mass of neoplastic tissue arising

Fig. 2.—Lateral roentgenogram of the skull, showing the shallow sella turcica and erosion of the anterior and posterior clinoid processes.
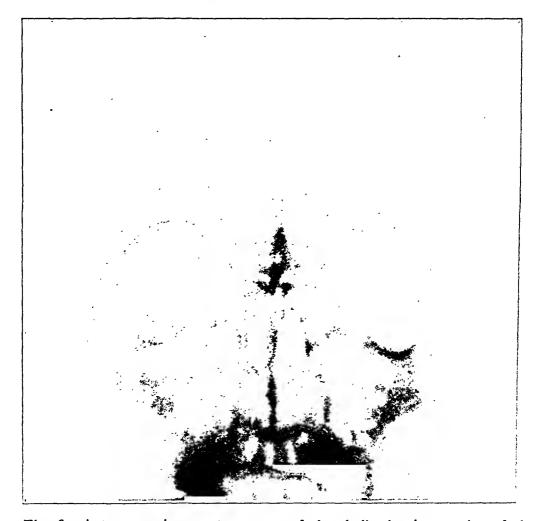
Fig. 3.—Anteroposterior roentgenogram of the skull, showing erosion of the left sphenoid ridge.

apparently in situ. The tissue was brown and yellow, with small cystic areas. The neoplastic optic nerve at the point where it passed the optic foramen ,had eroded the surrounding bone, resulting in a friable and crumbling sphenoid ridge. As the optic nerve passed into the orbit it mushroomed out into a red, fleshy growth which occupied the retrobulbar space. The anterior and posterior clinoid processes were eroded. The right optic nerve was swollen to about twice the normal size and at its proximal portion was infiltrated with neoplastic tissue spreading from the chiasm. There appeared to be a thinning of the left ventricle due to pressure upward of the chiasmal mass.

Microscopic examination of the tumor disclosed a glioma of a very vascular nature.

SUMMARY

A case is reported of a white woman of 43 years with a history of slow loss of vision of the left eye of many years' duration, progressing to complete blindness, and of exophthalmos and ptosis of the same eye, of recent onset. The positive findings were divergent strabismus and primary atrophy of the optic nerve of the left eye. The visual fields of the right eye showed temporal hemiachromatopsia for red and green. There were no general signs. Roentgenograms of the skull revealed a shallow sella turcica, erosion of the anterior and posterior clinoid processes, erosion of the left sphenoid ridge and enlargement of the left optic foramen. Operation disclosed a massive chiasmal tumor. Postmortem examination showed a tumor apparently arising from the chiasm and spreading laterally to involve both optic nerves and on the left side extending into the orbit, with resultant forward displacement of the globe.

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TREATMENT OF CAROTID ARTERY-CAVERNOUS SINUS FISTULA

REPORT OF A CASE

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BROOKLYN

In 1933 Hamby and Gardner [1] reported their observations following several surgical procedures which they employed in an attempt to bring about cure in two cases of fistula between the carotid artery and the cavernous sinus. In their case of significance in the present discussion (case 2) the patient had, in the order given, ipsilateral ligation of the extracranial portion of the internal carotid artery and the common carotid artery, ligation of the intracranial portion of the internal carotid artery distal to the lesion and finally ligation of the extracranial portion of the internal carotid artery and the common artery contralateral to the fistula. Two years later Dandy [2] reported the results following ligation (clipping) of the intracranial portion of the internal carotid artery in two patients who had a traumatic fistula between the cavernous sinus and the internal carotid artery. In each of these two instances there had been ligation of the extracranial portion of the internal carotid artery prior to the "clipping" of the intracranial portion of this artery. The reported results in these three cases indicate that ligation of the intracranial portion of the internal carotid artery (distal ligation of Brasdor) is applicable in those instances in which the intradural portion of the artery is not implicated in the pathologic process.

The following report illustrates the inadvisability of ligating the intracranial portion of the internal carotid artery in certain cases and describes an alternative for occluding a carotid artery-cavernous sinus fistula.

REPORT OF CASE

A 33 year old man was admitted to the hospital complaining of headache, vomiting and failing vision. When 7 years of age he was said to have fallen from a moving wagon and to have struck the occipital region of his head, which produced immediate unconsciousness. There was bleeding from the nose and ears, and the

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1. Hamby, Wallace B., and Gardner, W. James: Treatment of Pulsating Exophthalmos, with Report of Two Cases, Arch. Surg. 27:676 (Oct.) 1933.

2. Dandy, Walter E.: Treatment of Carotid Cavernous Arteriovenous Aneurysm, Ann. Surg. 102:916, 1935.

right eyelids were markedly swollen. After the return of consciousness, at the end of four days, it was noted that the left side of the face was paralyzed. He slowly recovered from this facial paralysis, but not completely so, a "difference" remaining between the two sides of the face. The swelling of the right eyelids subsided, but the eyeball on this side remained more prominent than its fellow. This protrusion of the right ocular bulb was noted to be more striking whenever he was excited or angry. He attended school until 16 years of age but was unable to finish the grammar grades. At 15 his vision was found to be defective, and since this time he had worn glasses. He had never been steadily employed, but worked at odd jobs as a handy man. As long as he could remember there had been a roaring noise in the head, sometimes loud enough to disturb his sleep. Difficulty in hearing had been present since the accident.

Although there had been occasional headaches all his life, in October 1935 he began to experience severe shooting pains across the frontal region of his head, which were more marked on the right side than on the left. These attacks of pain in the frontal region increased in frequency and on several occasions were associated with nausea and vomiting. Although his vision became progressively less acute during this time, the right eye did not become more prominent, and there was no appreciable change in either the character or the intensity of the noise in the head. No bleeding from the nose was reported. The pain in the head, vomiting and failing vision slowly became more marked, and he entered the hospital on Jan. 6, 1936.

Physical examination disclosed a sparsely built and poorly nourished man with an obvious defect of hearing. The right ocular bulb was prominent and displaced downward and outward (fig. 1). The lids of the eye, the right malar region and the right side of the forehead had a bluish tint due to the large and thickly set subcutaneous veins. The bulbar conjunctiva of the right eye was mildly chemotic, and the vessels of its mesial half were prominent. There was a visible as well as a palpable forward thrust of the affected eye at each cardiac systole. On palpation the large blood vessels that coursed vertically across the right side of the forehead were noted to have walls of such thickness that one gained the impression that the frontal bone was grooved. Obliteration of the flow of blood in these vessels by external pressure, first at the eyebrow and then at the hair line, indicated that the blood flowed from the orbital zone upward. A thrill with systolic intensification could be felt at any point about the right orbital area but was most striking at the inner aspect of the right upper lid. A soft, easily compressed prominence extended across the base of the nose. This seemed to be a large blood vessel communicating with the vessels of the medial aspect of the left orbital area. On auscultation over any part of the head a loud continuous bruit with a systolic accentuation could be heard. It was impossible to choose between the area about the base of the nose and the right temple as the point where this murmur was heard with maximum intensity. No musical quality could be detected, even after moderate exercise. Attempts to press the right globe into a normal position caused pain, although no great resistance was offered by the intra-orbital structures. The pupils were equal and reacted promptly to light and in accomodation. The optic nerve heads were sharply outlined and showed a mild degree of pallor. The veins of the right optic fundus were mildly engorged as compared with those of the left fundus. The visual fields showed marked concentric constriction. No scotomas could be demonstrated. The extra-ocular movements were not impaired, except for upward gaze, which was limited on the right side to the horizontal level. Strong attempts to gaze upward caused the right ocular

bulb to rotate inward. The fifth cranial nerves were intact. Very slight paresis of the left facial muscles could be demonstrated. Hearing was impaired, and this defect was shown by tests to be a lesion affecting conduction bilaterally. The remainder of the cranial nerves were considered normal. The neck was thin, and the pulsations of the right common carotid artery were more marked than those of the left. On palpation the right common carotid artery seemed to be about twice the size of the artery on the left side. Digital compression of this artery produced no change in the cranial bruit. The radial pulse rates were equal, and the blood pressure was equal in the two arms, the systolic pressure being 104 mm. of mercury and the diastolic pressure 48 mm. The heart was found to be small on physical and roentgen examination. The deep and superficial reflexes were present and were equal on the two sides. No sensory changes could be demonstrated.

Fig. 1.—Photograph of the patient taken on his admission to the hospital, showing the prominently displaced right optic bulb.

The gait and the equilibrium were within normal limits. Except for mild secondary anemia, all the laboratory examinations showed normal findings. The exophthalmometer readings were: 113 mm. at the base line, 27 mm. for the right eye and 12 mm. for the left eye.

On Jan. 13, 1936, with the patient under anesthesia induced with tribromethanol in amylene hydrate, 125 cc. of clear cerebrospinal fluid was removed fractionally by lumbar puncture and an equal quantity of air introduced. Roentgenograms of the head taken immediately after the introduction of the air showed a normally placed cerebral ventricular system. The entire system was moderately dilated, with the exception of the fourth ventricle, which was considered to be within normal limits. The right lateral ventricle showed a greater degree of dilatation than the corresponding ventricle of the left cerebral hemisphere (fig. 2).

On January 18, with the area under local anesthesia, the right common carotid artery and its two branches (the internal and the external carotid artery) were widely exposed and isolated. The lumen of the common carotid artery was completely occluded by a broad tape placed externally, and this occlusion was maintained for ten minutes. No abnormal signs or symptoms were observed during this period. Neither the size nor the tension (as noted on palpation) of the internal carotid artery was altered during this procedure, nor was the bruit in the head altered. Without removal of the occluding tape from the common carotid artery, a similar tape was placed around the right internal carotid artery, and its lumen was completely occluded. The patient was carefully observed (in regard to the fundi, the pulse rate, the blood pressure, respiration and the motor and sensory functions of the left extremities) for fifteen minutes; at the end of this period no change had been noted except a 40 to 50 per cent reduction in the intensity of the bruit as heard in the temporal fossae. Thereupon the internal carotid artery was doubly ligated with a silk suture, and the compressing tape was removed
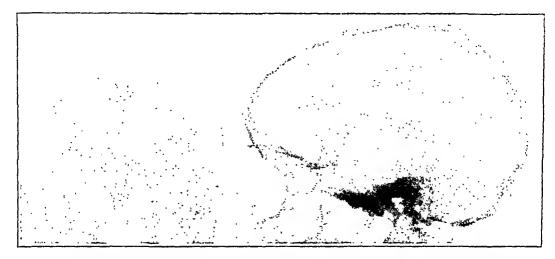
Fig. 2.—Anteroposterior and lateral views of the skull taken after the introduction of air by the lumbar route. In the anteroposterior view the general ventricular dilatation can be seen; the right lateral cerebral ventricle is larger than the left.

from the common carotid artery. Hemostasis was completed, and the wound was closed with a silk suture.

There were no untoward signs or symptoms following this operation. Two days after operation the right eye was less prominent but was still moderately proptosed and displaced (fig. 3). The intracranial bruit was considerably reduced in intensity but was easily heard with a stethoscope over all parts of the head. The thrill, which was palpable at about the inner aspect of the right orbit, was present. The patient was allowed out of bed on the fourth postoperative day. Although there was some improvement, in that the right ocular bulb had receded slightly and the intracranial murmur had become less intense, the visual fields showed little change. It was roughly estimated by these signs that the blood flowing through the arteriovenous fistula had not been reduced more than 25 to 30 per cent. After a five week period of observation, the intracranial bruit, as compared with that present immediately after the ligation of the internal carotid artery, had become more intense. At this time the exophthalmometer readings were: 113 mm. at the base line, 27 mm. for the right eye and 14 mm. for the left eye. Since the

ligation of the extracranial portion of the internal carotid artery had not resulted in satisfactory amelioration of the symptoms and signs, it was decided to ligate the intracranial portion of this artery, as advocated by Hamby and Gardner.

On March 3, with the patient under anesthesia induced with tribromethanol in amylene hydrate and the part under local anesthesia, the infundibulochiasmal area was exposed through a flap in the right frontal bone. An unexpected and unusual condition was observed. A vein fully 5 to 6 mm. in diameter ran parallel to the right lateral aspect of the optic chiasm and extended from the right optic foramen to a point level with the communication of the great petrosal vein with the cavernous sinus. There it crossed the right optic tract and entered the dural covering of what seemed to be the posterior aspect of the cavernous sinus. A second abnormal vein about 3 mm. in diameter coursed from the right optic
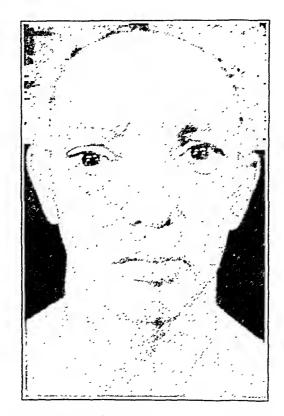
Fig. 3.—Appearance of the patient two days after ligation of the cervical portion of the right internal carotid artery.

foramen across the anterior aspect of the optic chiasm and entered the left optic foramen just medial and superior to the left optic nerve (fig. 4). Both these veins showed pulsations which were synchronous with those of the arteries exposed in the operative field. The arachnoid was moderately thickened about the entire chiasmal area. This was removed from the right side, and the right optic nerve, chiasm and optic tract, with the accompanying vein, were retracted mesially. In the usual position for the intracranial portion of the internal carotid artery there was an elongated, slightly domed pulsating surface, partial collapse of which could be produced by lateral compression over the cavernous sinus. From the distal part of this structure the internal carotid artery emerged and divided immediately. There was not space enough between the point of emergence of the internal carotid artery and its bifurcation (into the anterior and the middle cerebral artery) to permit application of a compression clip without partially or completely occluding
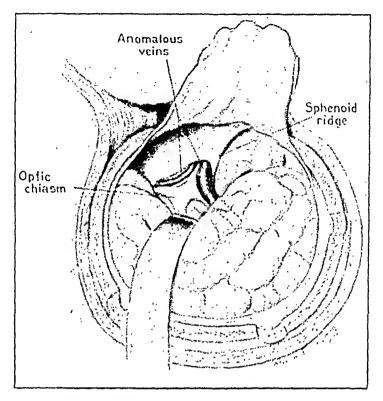
Fig. 4.—An artist's elaboration of the operator's sketch, showing the enlarged anomalous veins about the chiasm, as described in the text.

Fig. 5.—Appearance of the patient twenty days after the intracranial operation.

either or both of these branches (the right posterior communicating artery was not visualized). The hope of reducing the blood supply to the arteriovenous fistula by ligating the intracranial portion of the internal carotid artery was of necessity abandoned. It was discovered that on lateral compression of the area of the right cavernous sinus there was almost complete collapse of the two pulsating veins previously described. It was assumed that such compression mechanically blocked the fistula and that the collapsible mass occupying the position of the intracranial part of the carotid artery could be opened without causing excessive hemorrhage. A piece of temporal muscle approximately 1.5 cm. square and of the thickness of this muscle was cut, and when every detail was made ready the dome of the presenting mass was opened. The muscle was immediately introduced into the cavity, which it filled almost completely. The lateral compression was slowly released, and moderate pressure was applied over the small incision opening. After a period of waiting for maximum coagulation, the borders of the incision opening were fused with the enclosed muscle plug by the electro-coagulation unit. It was observed that the abnormal pulsating veins shown in figure 4 remained partially collapsed. After hemostasis of the wound, the bone flap was wired and the scalp closed with layer silk. Auscultation of the head while the patient was still on the operating table failed to disclose a murmur. The right pupil was widely dilated at this time, whereas the left was in middilatation.

The patient recovered satisfactorily from this procedure, and at no time during the postoperative course did he show evidence of any disturbance in the left extremities. The dilated right pupil returned to the size of its fellow in three days. There was marked improvement in vision. The condition of the eyes is shown in figure 5; this photograph was taken twenty days after the second operation. The prominence of the right eye became progressively less over a six month period, since which time it has remained about the same. The cranial bruit is not audible over any part of the head. The result at the end of one year seems entirely satisfactory.

COMMENT

As was stated in this patient's history, the traumatic carotid artery-cavernous sinus fistula on the right side was sustained when he was 7 years of age, and, as far as could be determined, the symptoms, aside from the evident intellectual deficiency, remained stationary for some twenty-five years. The fact that he had the lesion throughout most of the period of his physical development may account in part for the unusual anatomic findings at the time of the intracranial operation. No adequate textbook account of the veins within and about the optic chiasm could be found that gave a description of vessels which occupy the positions of the enormously enlarged veins here illustrated. Neither have I been able to find in the fresh cadaver veins, however, minute, so placed anatomically that they might dilate in the presence of a long-standing carotid artery-cavernous sinus fistula. It seems logical to assume that direct pressure of these large veins on the optic nerve within the optic foramen is the explanation of the visual disturbance in this case.

The slight general cerebral ventricular dilatation shown by the encephalogram may not be explicable on the basis of disturbance of

the blood supply to the brain; however, the fact that the right lateral ventricle was larger than the left lateral ventricle strongly supports the idea that the right cerebral hemisphere, as might be expected, had been deprived of a normal blood supply. The inability of this patient to advance in school with persons of his age group might also be attributed to the disturbed cerebral vascular condition.

Although direct occlusion of an arteriovenous fistula, such as was carried out in the instance herein reported, is the operation of choice, the technical safety of such a procedure is open to question. The occlusion of vascular channels by electrocoagulation is not as dependable as that accomplished by mechanical ligatures; yet this method is becoming more and more popular as a hemostatic agent in intracranial surgical procedures.

It would seem that a patient with a carotid artery-cavernous sinus fistula which is producing disturbing symptoms should be treated as follows:

1. Repeated digital compression of the affected internal carotid artery in the neck, if such compression produces cerebral symptoms and signs, may be helpful.

2. Mechanical occlusion of the common carotid and the internal carotid artery on the involved side, after they have been exposed, with the area under local anesthesia, may be carried out.

3. If these methods fail, ligation of the intracranial portion of the internal carotid artery just distal to the cavernous sinus should be performed.

4. If the intracranial portion of the affected internal carotid artery should prove to be implicated in the aneurysmal formation, the aneurysmal cavity may be opened and the fistula occluded with a piece of muscle.

Clinical Notes

A CASE OF OPACITIES OF THE VITREOUS OBSERVED FOR TWENTY YEARS AFTER SCLERO-CORNEAL TREPHINING

George Earle Henton, M.D., and George Herbert C. Henton, M.D.,
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On Nov. 2, 1916, the vision of the left eye of a 28 year old farmer was reduced to perception of light by a hemorrhage into the vitreous. On November 4 a complete survey, including a serologic study of the spinal fluid, gave negative results, but the teeth and tonsils were finally removed as suspicious. The vision of the left eye was limited strictly to perception of light for six months, after which time perception of light was lost permanently because of organization of the hemorrhage, and there was total detachment of the retina.

On March 4, 1917, a massive intravitreous hemorrhage in the "normal" right eye reduced the vision in this eye from 20/20 to perception of light. On June 18 a complete survey, including tests for sensitivity to tuberculin, carried out at the Mayo Clinic, gave negative results.

On August 7 the patient was seen by Dr. George E. Henton for the first time. The patient had been totally blind for six months, except for perception of light with the right eye, and he was much depressed. The vitreous of each eye was filled with débris, so no details of the fundus were visible.

For some time Dr. Henton had felt that a properly performed sclerocorneal trephining might facilitate elimination of vitreous opacities in carefully selected cases. It seemed advisable to apply this somewhat experimental procedure in this particular case, since the course in the right eye was apparently following that in the totally blind left eye, with no known treatment available. This was thoroughly explained to the patient and his family, and they unanimously requested that the operation be performed. With this understanding Dr. Henton performed a sclerocorneal trephining operation on the right eye on September 2. The vitreous slowly cleared after this operation, and after March 1918 the vision of the right eye was always 20/30 plus, without glasses.

As the fundus became visible for the first time since the hemorrhage on March 4, 1917, the upper temporal artery was seen to be occluded at a point 2 disk diameters from the center of the disk. There was an area of fibrosis and pigmentation about twice the size of the disk at this point in the fundus, and from this point a membrane of fibrous tissue extended into the vitreous. It was evident that the hemorrhage into the vitreous had originated from the point of occlusion of the upper temporal artery of the right eye. There was marked beading of the retinal arteries, with arteriovenous notching.

On Feb. 19, 1937, Dr. F. A. Kiehle confirmed our findings with the following report:

I have today examined Mr. A. W. and find that his vision is as follows: in the right eye, 20/30 plus, and in the left eye, no perception of light. The right eye shows a sclerocorneal trephine wound with a small bleb in the 12 o'clock position. The media are clear; the pupillary reaction is normal; the vitreous shows a wide, distinct gray band attaching from the disk along the route of the upper temporal artery superiorly and to the lower nasal region inferiorly—evidently the result of an old hemorrhage. It is impossible to get a clear view of the interior structure of the left eye, probably on account of an old intraocular hemorrhage.

At this time general physical examination gave negative results, except that the basal metabolic rate was lowered, on the probable basis of cerebral arteriosclerosis.

Dr. William L. Benedict and Dr. Frederick A. Kiehle supplied data published in this case report.

720 Southwest Washington Street.

METASTATIC PANOPHTHALMITIS FROM PYOGENIC CUTANEOUS INFECTIONS

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Metastatic panophthalmitis occurs fairly frequently, and cases of this condition in association with pneumonia, tonsillitis, meningitis, cholecystitis, parotitis and other diseases have been reported. In 1930 I[1] reported in the ARCHIVES a case of metastatic panophthalmitis occurring in association with calculous pyonephrosis. In this case Bacillus coli was the offending organism. Recently I had the opportunity of writing the pathologic report on an eye enucleated for metastatic uveoscleritis in a case of pneumonia. This case was reported in the ARCHIVES by Dr. Hulka.[2] In both these papers a complete bibliography was given and need not be repeated here. During the past few months my associates and I have had occasion to see at the Mount Sinai Hospital two cases of this condition occurring in association with meningococcic meningitis. It is of interest to note that in all the cases of subacute bacterial endocarditis which we have observed in the medical wards there has been no occurrence of metastatic panophthalmitis. Apparently Streptococcus viridans, which is responsible for many embolic phenomena in various organs, including the eyes, does not

1. Levine, Joseph: Metastatic Bacillus Coli Panophthalmitis from Calculous Pyonephrosis, Arch. Ophth. 3:410 (April) 1930.

2. Hulka, Jaroslav H.: Metastatic Pneumococcic Uveoscleritis Following Pneumonia, Arch. Ophth. 17:127 (Jan.) 1937.

initiate a suppurative process but rather causes a hemorrhagic reaction. It is also possible that the emboli are aseptic.

In a thorough search of the literature I have not been able to find an instance of metastatic panophthalmitis following a pyogenic infection of the skin.

The first case of this condition which I shall report is that of an Italian man 48 years old, who was admitted to the surgical service of the Mount Sinai Hospital in October 1935 for treatment of a large carbuncle of the neck. The patient was known to have diabetes and had been receiving 30 units of insulin daily. On admission his blood sugar content was 225 mg. per hundred cubic centimeters, and his urine contained 2 per cent of sugar. The amount of insulin that he received was increased to 45 units daily, and the carbuncle was incised and drained. Healing was slow, and three days after the operation his left eye became red and painful and he complained of blurred vision. Examination showed marked circumcorneal injection, a cloudy aqueous, a small pupil and edema of the lids. He was able to count fingers at 5 feet (1.5 meters). Despite local treatment with atropine and hot applications a hypopyon developed the following day. The blood sugar content at this time was 150 mg. per hundred cubic centimeters, and the urine contained 0.5 per cent of sugar. The vision of the eye two days later was limited to bare perception of light, and the local signs had progressed. Pain was extremely severe, and evisceration was performed. A culture taken at the time of operation yielded Staphylococcus aureus, which was the organism obtained from culture of the carbuncle at the time of incision.

The second case is that of a white man 60 years old who had had no history of ocular disturbance. His previous history was irrelevant. Two weeks prior to his admission to the hospital a rather large furuncle developed near the left nostril, with subsequent redness and swelling of the left side of the face. This swelling subsided in a week, but the furuncle was not entirely healed. The following day he noticed some impairment of vision of the right eye; the lids became edematous, and there was pain. Within two days the vision was reduced to perception of light, the eye bulged, pain was excruciating, and the edema increased. The family physician diagnosed the condition as acute glaucoma and referred the patient to the Mount Sinai Hospital. On his admission we found a healing furuncle of the left nostril. The right bulbar conjunctiva was chemotic and edematous; the eye could not be rotated and was proptosed directly forward; the lids were red and swollen; the pupil was extremely small, and the iris was bound down by exudate; the aqueous was cloudy, and the fundus could not be seen. The vision was sufficient to permit perception of movements of the hand. The patient's general condition, together with the local ocular signs and the history of loss of vision prior to the onset of proptosis and swelling, ruled out thrombosis of the cavernous sinus and orbital abscess. A culture of the blood gave negative results, but a culture taken from the eye at the time of evisceration of the globe revealed Staphylococcus aureus. Subsequent to the operation healing was uneventful and the patient was entirely comfortable.

The purulent emboli which come from the original seat of inflammation get into the general circulation, after which they enter the eye either through the central retinal artery, which is a branch of the ophthalmic artery, or through the short and long ciliary arteries, which are also branches of the ophthalmic artery. Regardless of which route (retinal or choroidal) is used, the outcome is the same. A deep-seated

intra-ocular purulent inflammation arises, which necessitates eviscera-
tion of the eyeball. It is usually advised not to enucleate such an eye,
because in enucleation the optic nerve and its sheaths must be cut,
and it is feared that some of the purulent contents of the globe may
have spilled onto the conjunctiva and thus be carried back with the
enucleation scissors into the optic nerve sheaths, to pass from there
into the cranial cavity and cause purulent meningitis. However, such
a condition can occur only if there has been perforation of the globe,
corneal or scleral, or both, with resultant spilling of the purulent con-
tents. Otherwise, infection is not to be feared, and enucleation is as
safe as evisceration.

Ophthalmologic Review

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PATHOGENESIS OF UNILATERAL EXOPHTHALMOS

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Unilateral exophthalmos is not of necessity similar etiologically to the bilateral condition. There is no doubt that many unilateral exophthalmic conditions may be simply incomplete, in terms of bilateral involvement seen in cases of exophthalmic goiter. It is just as true, however, that in many cases the primary characteristic is unilateral displacement of the eyeball. Involvement of the opposite side would be in such cases secondary and not consequent. Perhaps the best example of such instances is a case of neurofibromatosis.

It seems as though unilateral exophthalmos is properly considered a definite clinical entity in a large number of instances. Even in cases of unilateral exophthalmos associated with exophthalmic goiter this condition, in my experience, presents various signs less commonly seen in cases of the bilateral variety. There is no certain reason why exophthalmos should at times be almost purely unilateral, but the fact stands that cases of this condition do occur, and the disorder need not become bilateral at some later stage. Differences in the degree of exophthalmos in both eyes, when this is gross, may account for some cases of unilateral exophthalmos, especially those in which equal exophthalmos develops later in the progress of the toxic state.

The anatomic reasons for exophthalmos are not complex.

Wolff,[1] in his description of the orbit, spoke of it as a goblet-shaped cone open in front, within which, posterior to the eyeball, there is a closed fasciomuscular space. Eagleton[2] said: "Overfilling of this space, even from slight circulatory disturbances, which in other parts of the

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1. Wolff, E.: The Anatomy of the Eye and Orbit, London, H. K. Lewis & Co., Ltd., 1933, p. 310.

2. Eagleton, W. P.: Exophthalmos from Surgical Diseases, Especially as to Involvement of the Protective Retrobulbar Space, Arch. Ophth. **14**:1 (July) 1935.

body gives rise to flushing and congestion, as in hyperthyroidism, here causes exophthalmos." The orbit is of mesoblastic origin, filled with elements of ectodermal and mesodermal origin and formed of rigid walls from the same embryonal source; and it contains highly specialized neural tissues from the fetal forebrain. Adult dural elements appear in its apex, and through these a somewhat active connection is present with the intracranial cavity. Further, it is almost wholly without lymphatic tissue, and its veins are without valves. The venous supply is unusually copious, considering its position, and it is closely connected with the venous drainage of the face and of the nasal cavity and with the nasal accessory sinuses emptying into the cavernous sinus and into the pterygoid plexus of veins. The architecture of its bony structure, while favorable to the integrity of the eyeball and its important adnexa, is by reason of its own rigidity a dangerous cul-de-sac for a malignant process and acute infection and for some chronic inflammatory processes. A pathologic condition of the orbit is seldom a cleancut medical or surgical condition from a diagnostic standpoint. More often it is a problem the solution of which taxes every bit of one's diagnostic acumen.

The cases of unilateral exophthalmos can be divided into several different groups from the standpoint of etiology. Such a grouping would include cases of unilateral exophthalmos due to: anatomic conditions—including extra-orbital pathologic disorders—some with no apparent orbital pathologic changes; traumatic conditions; acute, subacute and chronic inflammatory conditions; conditions of the blood-forming and lymphatic systems, and neoplastic diseases. The last group includes nonparasitic cysts as well, even though they are not neoplasms. Space-taking lesions would be, perhaps, a better term for this group. The diseases of the lymph and the hematopoietic system should be considered separately in that, while they have a common anatomic origin, they are not commonly considered in a discussion of the formation of tumor cells.

The following classification is suggested, with the hope that it is a fairly complete one.

Anatomic Conditions.

1. Unilateral myopia of a high degree.
2. Defects in the vault of the orbit; meningocele and meningo-encephalocele.
3. Exophthalmos associated with ocular hypertension.
4. Exophthalmos associated with arterial hypertension.
5. Pseudo-exophthalmos resulting from retraction of the lids.
6. Intermittent exophthalmos.
7. Disease of the pituitary gland.
8. Exophthalmos resulting from irritation of the cervical portion of the sympathetic nervous system.
9. Unilateral exophthalmos associated with toxic goiter.

Traumatic Conditions.

1. Fracture of the orbit (with retrobulbar hemorrhage).
2. Laceration and rupture of the extra-ocular muscles.
3. Traumatic evulsion of the globe.
4. Trauma sustained at birth—intracranial; aneurysm of the internal carotid artery.
5. Foreign body in the orbit.
6. Pulsating exophthalmos; arteriovenous aneurysm.
7. Spontaneous retrobulbar hemorrhage, as from whooping cough.

Inflammatory Conditions.

These must be acute, subacute or chronic.
They are given here roughly in the order of their chronicity.

1. Retrobulbar cellulitis.
2. Retrobulbar phlegmon.
3. Retrobulbar abscess.
4. Thrombophlebitis of the orbital veins.
5. Thrombosis of the cavernous sinus.
6. Erysipelas.
7. Tenonitis (at times suppurative).
8. Periostitis (syphilitic or tuberculous, but not including gumma; pseudo-tumor; disease of the lacrimal sac).
9. Orbital mucocele; pyocele; cholesteatoma.
10. Orbital exostosis.
11. Paget's disease, with hyperostosis.
12. Actinomycosis; trichinosis; mycotic pseudotumor.
13. Xanthomatosis allied to the Schüller-Christian syndrome.

Diseases of the Blood, the Lymph and the Hematopoietic System.

1. Rickets; scurvy; hemophilia.
2. Lymphosarcoma (Piney; lymphoblastic).
3. Acute and chronic lymphatic leukemia; lympho-endothelioma; Mickulicz' disease (Wolff; malignant lymphoma).
4. Hodgkin's disease (a sclerosing type of lymphoma).
5. Myeloblastoma; chloroma (a bone marrow picture).
6. Myelogenous leukemia (premyelocytes and myelocytes).

Space-Taking Orbital Lesions.

The order here is, roughly, the degree of malignancy, as seen and reported by many different observers.

1. Pseudotumor.
2. Dermoid cyst; sebaceous cyst; gumma.
3. Fibroma.
4. Neurofibroma.
5. Lipoma.
6. Osteoma; osteofibroma.
7. Myxoma.
8. Chondroma and chondromyxoma.
9. Cylindroma of the lacrimal gland.
10. Psammoma.

11. Adamantinoma.
12. Lymphangioma and hemangioma.
13. Plasmoma.
14. Meningioma or endothelioma.
15. Rhabdomyoma.
16. Tumor of glial tissue, such as neuroma, neuroblastoma, retinoblastoma and spongioblastoma polare.
17. Tumor of the sphenoid ridge.
18. Carcinoma.
19. Sarcoma; mixed cell tumor; intra-ocular sarcoma.
20. A malignant growth extending from the nasal accessory sinuses (including carcinoma, sarcoma, psammoma, chondroma and myxoma).
21. A metastatic malignant growth, such as osteogenetic sarcoma, adeno-carcinoma and hypernephroma.

ANATOMIC CONDITIONS

Axial myopia, unilateral and of high degree, is not uncommon, and this condition occurs frequently in connection with divergent strabismus as well. The anteroposterior axis of the eyeball attempts to adjust itself to the axis of the orbit. Further, one can see in some of these cases a slight but definite increase in the exophthalmos when the patient rotates his eye toward the contralateral side. The condition in the case which is cited here is representative. The outstanding characteristic was that the parents brought the patient to the ophthalmologist because of the "bulging of the right eye" and not because they were aware of a failure in visual acuity. The patient was the 10 year old son of an army officer. He showed extropia of 15 degrees for both distant vision and near vision, exophthalmos of 9 mm. when looking straight to the front and an increase in this of about 2 mm. when looking toward the left. Central visual acuity was 1/400. There was axial myopia of —18 D., associated with a pathologic condition of the fundus which showed plainly extensive recent progression in the myopia, i. e., macular choroidal sclerosis and a huge peripapillary staphyloma. Examination of the left eye showed a slight degree of hyperopic astigmatism.

Defects in the vault of the orbit may be present and the eye may not show any exophthalmos. Routine roentgen studies of the orbit prove this. These dehiscences are rather commonly connected with the position of the normal sutures. Meningocele, meningo-encephalocele and encephalocystocele have all been reported as causing exophthalmos because of such congenital anatomic defects. Safranek[3] reported a case in which recovery followed surgical treatment. Edson and others have corrected such conditions by operations in which they used a transfrontal approach and implanted bone grafts on the roof of the orbit. A roentgen

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3. Safranek, J.: Operierter Fall einer Cephalocele naso-orbitalis. Ztschr. f. Hals-, Nasen- u. Ohrenh. **3**:560, 1922.

examination will be sufficient for the diagnosis in such conditions. In most of the cases the protruding substance enters the orbit through the medial wall.

Figure 1 illustrates a case of meningo-encephalocele observed by Seefelder, of Innsbruck, Austria, and reported by Atkinson. It shows well the presentation through the medial wall, which is common in this condition.

Exophthalmos is occasionally seen, associated with arterial hypertension and with unilateral ocular hypertension, the exophthalmos in both instances being unilateral, without any orbital cause for the proptosis being demonstrable. One patient of Peter's with such a condition was operated on. The operation consisted of approach by a transfrontal route to the anterior fossa, removal of the orbital roof (orbital decompression) and thorough exploration of the orbital contents for the detection of any possible pathologic condition. None was found except

Fig. 1.—Meningo-encephalocele (case of Prof. R. Seefelder, Innsbruck, Austria; from Atkinson, D. T.: External Diseases of the Eye, Philadelphia, Lea & Febiger, 1934; reproduced by permission of the publisher).

a possible increase in the blood supply of the orbital fat. The eyeball continued to show exophthalmos of the same degree and little, if any, difference occurred in the glaucomatous hypertension. Vision has been preserved by other methods. In those cases in which there is arterial hypertension (this was not present in the case just described), the hypertension may be explained on the basis of varicosities of orbital vessels. One patient with such a condition, an inmate of a home for old soldiers, was under observation for several years. He showed unilateral exophthalmos, with thick, moist, painless chemosis of the inferior conjunctival cul-de-sac. Vision was only slightly impaired; this was due, undoubtedly, to the angiosclerotic condition of the retina. The patient was finally sent back to the home of some relatives, the condition having remained unchanged in all the details. The cause of his death is unknown, for nothing further has been heard about him.

Pseudo-exophthalmos is seen not uncommonly in association with various disturbances of cranial nerves. It is not a true exophthalmos. A slight difference may be present in the anteroposterior position of the eyeballs, but this would be familial and not connected with the existing condition. The resemblance to exophthalmos results from widening of the palpebral fissure due to retraction of the lids. At times it seems pronounced. Paralysis of the seventh cranial nerve when present for a long time causes this most frequently. Figure 2 illustrates this condition. The duration of the facial paralysis in this instance was eighteen months; vision was normal, and the patient was distressed only because of the cosmetic deformity.

Intermittent exophthalmos has been described by several observers. Eagleton[2] spoke of a case of this condition in which the exophthalmos was associated with suppurative otitis and recurred with each successive attack. He said:

As the ocular protrusion could also be produced by pressure over the right jugular vein, or by turning the head to the right, and as it persisted after the

Fig. 2.—Pseudo-exophthalmos of the right eye, associated with old facial paralysis.

aural suppuration had subsided, the intermittent exophthalmos was doubtless due, not to infection, but to a congenital anomaly of the vessels returning the venous flow from the orbit.

Posey[4] also reported a case of intermittent exophthalmos, in which the eyeball could be voluntarily proptosed. This case may have been one of a dehiscence of the orbital wall into the ethmoid labyrinth, though varicosities of vessels may equally well have been the cause.

Disease of the pituitary gland is occasionally connected with unilateral exophthalmos, without orbital invasion by a neoplasm. In the two cases of this condition observed by me roentgen examination showed no differences in the two optic foramens, nor were there any oculomotor disturbances to account for the exophthalmos. The condition may have developed from orbital venous stasis, but the veins of the fundus should have shared in this stasis, apparently, and certainly the superficial veins which drain into the cavernous sinus would have shown obstruction.

4. Posey, quoted by Shoemaker, W. T.: Disease of the Orbit, in Ball, J. M.: Modern Ophthalmology, ed. 3, Philadelphia, F. A. Davis Company, 1913.

In spite of the absence of these signs, it is rather probable and logical that venous stasis was the cause for this proptosis. Naffziger [5] stated that this is most common in the eosinophilic forms of adenoma of the pituitary gland.

Exophthalmos of a unilateral character has been reported as the result of irritation of the cervical portion of the sympathetic nervous system. In these cases the exophthalmos will be accompanied by widening of the palpebral fissure and mydriasis. The causes, as discovered, include disease of the cervical glands, cervical ribs, aneurysm of the large vessels of the neck and neoplasm of the cervical region. The opposite condition, paralysis of the cervical portion of the sympathetic nervous system, with its enophthalmos, should not confuse one and lead to a diagnosis of unilateral exophthalmos in the normal eye.

Unilateral exophthalmos resulting from, or associated with, toxic thyroid disease is most interesting. The bilateral form of exophthalmos associated with toxic thyroid disease is still unsatisfactorily explained from several standpoints. It may occur, first, in connection with an elevated basal metabolic rate and, second, when the basal metabolism is within the normal limits and when all other constitutional symptoms are absent. Zimmerman [6] spoke of this condition as paradoxical exophthalmos and rightly considered it the more serious disorder. Two cases of a severe form of this condition have been observed. In one patient, a one-eyed man, the eyeball, in spite of the several surgical procedures, was completely destroyed by corneal ulceration and through spontaneous rupture. The second of the patients had similar severe bilateral malignant exophthalmos, and there was so much corneal damage that enucleation of both eyes was recommended before the almost inevitable spontaneous rupture occurred. In these cases, as Plummer and Wilder [7] pointed out, the malignant exophthalmos is rather likely to be unilateral, "or if not unilateral, it is likely to affect the eyes unequally." The exophthalmos (bilateral), which occurs with the elevated metabolic rate, may be explained in part, in spite of much controversy, by the spastic contraction of Müller's orbital fibers acting against the pull of the weakened stripped muscle fibers of the four rectus muscles. The absence of mydriasis under these conditions may be accounted for by the observations of Labbé and his associates, as quoted by Plummer and Wilder, [7] that "thyroxin not only has a sympathomimetic action but it also stimulates

5. Naffziger, H. C.: Pathologic Changes in the Orbit in Progressive Exophthalmos, Arch. Ophth. **9**:12 (Jan.) 1933.

6. Zimmerman, L. M.: Exophthalmos Following Operation for Relief of Hyperthyroidism, Am. J. M. Sc. **178**:92 (July) 1929.

7. Plummer, W. A., and Wilder, R. M.: Constitutional Factors with Particular Reference to Exophthalmic Goiter, Tr. Am. Acad. Ophth. **39**:41, 1934.

the parasympathetic elements of the autonomic nervous system." One cannot be convinced by those theories which consider the cause to be an increase in the orbital contents.

In regard to the exophthalmos which occurs in patients who have a low basal metabolic rate Plummer and Wilder [7] made the following statements:

Why should the external rectus muscles become swollen and hardened, and enlarge to several times their normal thickness? Why should edema develop in the lids when it was absent in the stage featured by a high basal metabolic rate? The evidence from the laboratory suggests that overfunction of the anterior lobe of the pituitary body may play a part in the development of this abnormality. Another possibility is presented by the two-product hypothesis of Plummer,[8] which is based on the assumption that the abnormal product continues to act after the output of the normal product, thyroxin, has been curtailed. The edematous contents of the orbit in this variety of exophthalmos are comparable in some respects to localized subcutaneous areas of mucinous edema, which are found in rare cases of exophthalmic goiter, particularly after thyroidectomy.

Four different cases are sufficient to illustrate various other points in the unilateral form of exophthalmos associated with a high plus basal

Fig. 3.—Unilateral exophthalmos associated with acute toxic thyroid disease.

metabolic rate. The first case, which is illustrated by figure 3, is one of bilateral proptosis in a patient in an acute phase of thyrotoxicosis, but there was a marked difference in the amount of the exophthalmos present in the two eyes. The second case is one of an 11 year old child who was brought in for attention because of unilateral exophthalmos associated with hyperphoria and with diplopia. Her plus basal metabolic rate promptly revealed the true condition. The patient has been under further observation for five years, and the disease has followed the classic course of acute toxic goiter. The unilaterality of the exophthalmos has remained unchanged. The third case is one of long-standing unilateral exophthalmos in a man 45 years of age. He sought relief at the Peter Clinic because of paralysis of the superior rectus muscle and the external rectus muscle of the proptosed eye. Surgical attention corrected his diplopia, and he was advised to seek medical attention because of his

8. Plummer, H. S.: Functions of the Normal and Abnormal Thyroid Gland, in Christian, H. A., and Mackenzie, J.: Oxford Medicine, New York, Oxford University Press, 1920, vol. 3, pp. 846 and 973.

moderately increased basal metabolic rate. At the same time he was under the care of a neurologist, who made a diagnosis of psychoneurosis. The patient was seen at various intervals over a period of three years. Up to his departure from the city he had not shown any great changes, though the constitutional signs of toxic goiter increased at various times. The fourth case is one in which there was a low basal metabolic rate associated with a moderate degree of bilateral exophthalmos (which might ordinarily escape attention), in which dissimilarity was evident in the degree of exophthalmos in the two eyes. Figure 4 illustrates the fourth case. In patients with this condition, also, there is imbalance of the ocular muscles, which seems to be a constant accompaniment of the unilateral form of exophthalmos associated with toxic thyroid disease.

Relative to the microscopic anatomic features of the orbital contents in exophthalmos associated with thyroid disease, various features have been reported which cannot be characteristic of the thyrotoxicosis. There is only one certain fact about these—as Naffziger has pointed out,

Fig. 4.—Bilateral exophthalmos, which is dissimilar in the two eyes, associated with toxic goiter.

"The microscopic pictures of the orbital muscles in myasthenia gravis and in progressive exophthalmos associated with thyroid disease cannot be differentiated one from the other; they are identical." It is unfortunate that while one must acknowledge the presence and the etiology of this condition as established facts, one also must confess to knowing little, if anything, as to the true reasons for the exophthalmos.

TRAUMATIC CONDITIONS

The traumatisms which cause unilateral exophthalmos are not unusual or uncommon, with one exception, one of the last to be presented herein. A history of trauma, emphysema, exophthalmos and impaired ocular motility and the roentgen findings are sufficient for a correct diagnosis in all cases. One must not be misled, however, by a history of a slight degree of causative trauma. Extensive fractures have followed relatively minor accidents, and, conversely, severe traumas have frequently resulted in but few pathologic changes in bone. A case was

reported by Peter [9] in which the injury involved all the ocular muscles except the superior oblique muscle, the trauma being a simple pen-knife stab wound of the orbit, and the only fracture present was a penetrating wound of the lamina papyracea ethmoidalis. (The later history in this case, incidentally, illustrates the reconstructive work and satisfactory results possible in such instances.) Evulsion of the optic nerve and the usual exophthalmos resulting from laceration of the extra-ocular muscles occur from slightly different accidents. In a case of the first mentioned of these two, the condition resulted from a blow to the eye from a broom handle which had been thrown as one would hurl a javelin. The ophthalmoscopic picture and the sudden complete blindness which occurred confirmed the diagnosis (fig. 5). The second of the aforementioned conditions is illustrated by 2 cases in which the injury was rather similar; in the first a child fell on the spout of an oil can, and in the second a boy, somewhat older, fell on the top of a steel picket fence while climbing this during play. The crepitus and the emphysema which accompanied the condition in this case also illustrate findings usually present whenever an orbital fracture is compound, involving the

Fig. 5.—Old exophthalmos of the left eye, with divergence, resulting from evulsion of the optic nerve.

nose and especially a nasal accessory sinus. In all these cases the orbital hemorrhage which occurs is likely to be the greatest factor in producing the exophthalmos which immediately develops. Every one who has seen exophthalmos resulting from retrobulbar hemorrhage following the injection of an anesthetic into the retrobulbar space prior to surgical intervention for cataract will agree to this probability.

Trauma sustained at birth can be serious. The injuries which result from the application of obstetric forceps during a difficult delivery are, fortunately, usually temporary. Occasionally the effects of such traumas are permanent. The cranial bones of the vault, when subjected to various different forms of injury, such as contusions, a penetrating wound and even crushing injuries, react with lines of fracture which follow patterns approximately common for all similar injuries. Holloway, as well as various anatomists, demonstrated this. This traumatic factor is simplified, decidedly, it seems, when orbital fractures are considered. The heavy wedge of the zygomatic arch and the curve of the upper

9. Peter, L. C.: Tr. Am. Ophth. Soc. **31**:232, 1933.

outer angle of the frontal arch ordinarily prevent injury to the orbital surface of the greater wing of the sphenoid bone, unless they are themselves fractured. If they are fractured, the sphenoid bone must be injured as well. The floor of the orbit is a rather thin partition separating the orbit from the antrum and continuing medially with the os planum of the ethmoid bone (bone structures similar in density and in structure) and is rather readily fractured from direct blows to the face and to the region of the orbit. The medial wall, at the apex of the orbit, may be included in these lines of fracture. In such instances the fracture is almost certain to continue into the bones at the base of the skull, perhaps into the temporal bone and, when it extends through the sphenoid bone, into the basilar process of the occipital bone. Figure 6 shows such an injury, which resulted from trauma caused by an obstetric forceps, involving the rim of the orbit, with the formation of an encapsulated sequestrum there and with a fracture through the
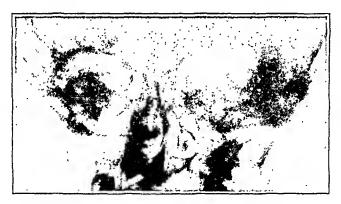
Fig. 6.—Roentgenogram of the orbit, showing the results of trauma sustained at birth and old fractures of the floor and the outer wall of the orbit.

sphenoid bone into the base of the skull. Additional pathologic changes were a fibrohemangioma of the orbit (removed through a Krönlein resection) and a pulsating intracranial aneurysm of the internal carotid artery which had eroded the right side of the tuberculum sellae and the right clinoid processes. A roentgenogram showed this well (fig. 7 A). The pulsating mass was felt in the orbit at the time of the resection of the orbital wall, and following the removal of the subepithelial and orbital hemangioma the pulsation of the intracranial aneurysm was transmitted to the eyeball to a remarkable degree. This pulsation had been present before the resection of the orbital wall, and its cause was correctly determined by both the ophthalmologist and the roentgenologist, but it was startling to see the tremendous increase in the pulsation immediately after the operation. No bruit was present at any time; the fundus showed no pathologic changes, and the fields and central visual acuity were entirely normal. According to Grant, the prognosis in these cases is often grave. Transfrontal craniotomy and/or fractional

ligation of the internal and the common carotid artery done separately or together must be considered. Recently some success has been achieved with high voltage roentgen therapy.

An aneurysm of the internal carotid artery in an adult was diagnosed post mortem by Groff in the case of a patient with left unilateral exophthalmos who was admitted in a semicoma to the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases. The blood-tinged spinal fluid was under a pressure of 500 mm. of water. There were no oculomotor disturbances. A swelling of the left nerve head, measuring from 12 to 14 D., was present when the patient was first seen by Groff and me. This involved the entire retina, giving a picture not unlike that of massive retinal detachment. Death followed shortly. The antemortem diagnosis was intracranial hemorrhage either from a ruptured aneurysm
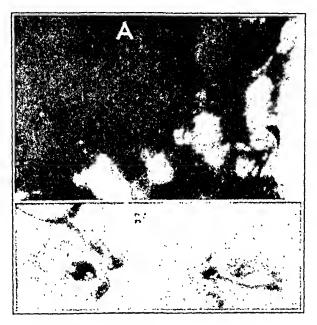
Fig. 7.—*A*, roentgenogram showing the sella turcica in a case of pulsating intracranial aneurysm; *B*, photograph showing the condition in the same case illustrated in *A*, with the lids open. Hemangioma of the orbit is also present.

or due to a neoplasm, with subsequent erosion of one of the carotid arteries, one of the jugular bulbs or one of the cranial venous sinuses. It was thought that the pathologic condition of the fundus might be an extension of the hemorrhage along the sheath of the optic nerve into the eye and might not be due to intracranial pressure. The normal condition of the right fundus seemed to confirm this. Transillumination failed to show any opaque mass in the fundus. De Long's report of the microscopic examination of the eyeball which was enucleated at postmortem examination is as follows:

I am inclined to believe that we are dealing with a choked disk, the result of some intracranial pressure. This is verified by the dilatation of the intra-

vaginal spaces and the edema of the nerve head, with practically empty and compressed blood vessels. There is a questionable exudate associated with the choked disk, which has separated the retina from the choroid. In the postmortem specimen much of this exudate has been washed away, but remnants remain.

Pulsating exophthalmos, that is, exophthalmos due to an arteriovenous aneurysm, either of the carotid-jugular sinus or of the carotid-cavernous sinus, usually has a history of injury preceding the exophthalmos. The typical bruit is present; changes of the fundus are not uncommon, and the bruit, the exophthalmos and the pulsation can be modified by temporary pressure over the carotid artery in the neck. One case of this condition was that of a 26 year old soldier at the Walter Reed Hospital. Enucleation was done because of blindness resulting from accompanying serious corneal damage. This modified the bruit to some degree, but fractional vascular ligation was later necessary because of a late marked increase in this bruit. Peter saw a patient with pulsating exophthalmos without a history of injury—an obese woman of 40. Serologic tests were negative throughout. Fractional ligation was completed on the right and helped to some extent, but for a short time only. Secondary glaucoma, which became absolute, developed in the right eye. In this case the history, which yielded no record of trauma, may not have been accurate, but the patient was positive in her answers to all questions relative to this point. The results of surgical intervention on blood vessels in cases of this condition are often disappointing. Owing to later contralateral anastomoses, the pulsation and exophthalmos may recur. If death does not intervene, contralateral hemiplegia may develop as a result of cerebral softening, and the patient lives a miserable existence.

Relative to the diagnosis of these intracranial vascular conditions Møller[10] studied the records of 555 patients with aneurysm of a large cerebral vessel. Choked disk was not found in any of them. Oculomotor paralysis was common; bulbar protrusion was rare, and pulsation of the eyeball was never seen in the simple (not the arteriovenous) variety. This form was slightly more common in the female than in the male. The classic signs and symptoms of the arteriovenous type of aneurysm include, in addition to the aforementioned, a history of injury; pulsating exophthalmos, and pounding or ringing sensations in the head, i. e., the bruit. It is interesting that the findings in the 4 cases cited in this section should not agree wholly with these findings. Figure 8 illustrates a case of unilateral exophthalmos associated with an arteriovenous aneurysm in a patient of Eagleton's who showed exophthalmos of a high degree. The real problem in all these cases is the

10. Møller, P.: On Intracranial Carotid Aneurism, Klin. Monatsbl. f. Augenh. **66**:909, 1921.

differentiation of an orbital aneurysm from an intracranial aneurysm. Møller's findings must be considered seriously in such possible perplexities.

A foreign body in the orbit is not uncommon. The history is usually clearcut, and a roentgenogram will clear up any doubts which may exist. The history should also reveal the substance of the foreign body. Removal is not essential in the absence of infection or other possible complications, if the foreign body is of steel or iron. Foreign bodies of other materials, however, may cause late complications of a serious nature. Hence their removal may be necessary. Damage to the muscles is likely to be an accompaniment. A pathologic condition of the optic nerve may develop, if not present from the start. Experiences in military surgery have clarified decidedly the treatment of these conditions. Removal should not be attempted in a case of the usual type of this condition, however, without the employment of a biplane fluoroscope. The facts in one such instance satisfied me as to this

Fig. 8.—Unilateral exophthalmos associated with traumatic pulsating arterio-venous aneurysm (Eagleton's case).

when a second unsuccessful attempt was made to remove a piece of copper lying posteriorly and close to the medial wall of the orbit. In this case, however, the exophthalmos and diplopia were largely the result of complete paralysis of the third nerve.

Spontaneous hemorrhage, which was subperiosteal, with unilateral exophthalmos, following a paroxysm of coughing in whooping cough has been reported. A case of this condition was described by Peter.

INFLAMMATORY CONDITIONS

In these conditions the exophthalmos is usually confined to one orbit. It may, however, become bilateral. The exophthalmos may occur simultaneously in each eye, but usually there is quite an interval before the second eye becomes involved. I have seen this occur in a case in which there was an interval of several weeks before the second eye was involved, while the patient was still suffering from an attack of disease of the nasal accessory sinuses (acute or subacute).

In considering the routes through which inflammation can enter the orbit, Eagleton [2] is quoted. The routes are:

(*a*) from in front, by way of the superior ophthalmic plexus, (*b*) from below, from the pterygoid plexus or through the inferior ophthalmic plexus, or (*c*) medially, from the nose. Endophlebitis of the vessels within the space (*a*) may remain thrombophlebitis of one or the other of the ophthalmic veins . . . or it (*b*) may form a local abscess within the cone, although infection of the retrobulbar space (*c*) is usually but a part of a generalized orbital phlegmon . . A suppurative lesion of the space usually occasions (1) cavernous sinusitis with (2) terminal meningitis. However, the infection of the space itself may arise secondarily from cavernous sinus phlebitis.

Two extensive series of cases may be cited here to illustrate the incidence of inflammatory exophthalmos in cases of sinus disease. Cohen reported a series of 1,517 patients with sinus disease, in 22 of whom inflammatory exophthalmos developed. Birch-Hirschfeld reported on a series of 648 patients· with inflammatory exophthalmos; 409, or 60 per cent, showed sinus disease. Among these, the frontal sinus was involved in 30 per cent, the maxillary sinus in 22

Fig. 9.—Exophthalmos (pseudotumor) associated with disease of the ethmoid sinuses.

per cent, the ethmoid sinuses in 21 per cent and the sphenoid sinus in 6 per cent. Further, in most instances more than one sinus was involved.

Cellulitis and phlegmon do not always continue to the formation of an abscess. Figure 9 illustrates an instance in which no abscess formed. Thorough investigation of the patient's subperiosteal space failed to show any pus. After incision of the periorbita the prolapsed retrobulbar fat appeared bluish, and there was marked vascular dilatation. A hemolytic streptococcus was grown from the culture tubes. The ethmoid cells were broken into (at the same time), and drainage was established from the subperiosteal space to the surface of the skin and through the ethmoid cells into the nasal cavity. At the time of the operation there were: exophthalmos of more than 1 cm., complete immobility of the eyeball, and beginning papillitis in the same eye. For twenty-four additional hours the child continued to be gravely ill. Forty-eight hours later he was definitely on the road to recovery. At no time was there any manifest external drainage from the subperiosteal space; still it is certain that this decompression (if one wishes to call

it this) was the turning point in the child's progress. The signs resembled those of early unilateral thrombosis of the cavernous sinus. As to the diagnosis, the proptosis and immobility of the globe, the papillitis, the roentgen and clinical signs of acute disease of the ethmoid and frontal sinuses, a temperature of the serious septic type and the cytologic features of the blood characteristic of sepsis were conclusive evidence. The condition is somewhat different from that in which an orbital abscess develops early. The clinical signs, in such cases, are not as severe, and the exophthalmos develops rapidly. The changes of the lid are pronounced. Papillitis does occur, but uncommonly. Osteomyelitis of the orbital roof may be present early in cases of this disorder. The condition of a colored child, 15 years of age, illustrates this type of exophthalmos, as well as the salient features in the formation of an orbital abscess. Acute rhinitis was present for five days; the lids began to swell on the fifth day, and the eyeball began to protrude on the seventh day after the onset of the rhinitis. The temperature was septic and moderately elevated. The child was sick but not prostrated by the

Fig. 10.—Orbital abscess resulting from disease of the frontal sinus (case of Simpson, Memphis, Tenn.; from Atkinson, D. T.: External Diseases of the Eye, Philadelphia, Lea & Febiger, 1934; reproduced by permission of the publisher).

intoxication. The fundus was normal. Incision and drainage were done on the night of the eighth day, and the patient was discharged from the hospital as recovered eight days later. Figure 10 illustrates a case of orbital abscess which was cured by this treatment, a form of therapy which is, at the same time, diagnostic.

Exophthalmos associated with an abscess, which develops more slowly, and has even fewer signs of acute sepsis is likely to originate in disease of the frontal sinus and to be accompanied or perhaps caused by osteomyelitis of a frontal bone. An instance of such a condition is a 45 year old man with exophthalmos which had been slowly developing for five weeks and with but few signs or symptoms of acute sepsis. At orbital incision and drainage, an enormous amount of retrobulbar suppuration was found, with considerable osteomyelitic loss of the floor of the frontal sinus at the inner nasal angle. This patient died with generalized meningitis, and with an extradural abscess localized in the

frontoparietal region. In cases in which the frontal sinus is involved, the condition is always serious, and there is a high mortality rate.

Thrombophlebitis of the orbital vein and thrombosis of the cavernous sinus are preceded by orbital cellulitis and by abscess in a rather small percentage of cases. Disease of the nasal accessory sinuses is a frequent cause, but other conditions are as frequently the cause. A Negro of 45 showed the former of these conditions, with subsequent involvement of the cavernous sinus following the appearance of a small pustule at the outer canthal angle. He lived for four weeks, and at one time it was thought that he might recover. A private patient of Peter's died within forty-eight hours after the onset of the orbital signs of sinus thrombosis in the first eye, following dental packing after the extraction of an abscessed root of a tooth, which packing had been retained for twenty-four hours longer than the exodontist had advised.

In cases of classic disease of the cavernous sinus, thrombosis develops rather early in the infection and seems to be due usually to the involvement of the sphenoid sinus. The exophthalmos and the immobility on one side develop in this sequence and not slowly, and thereafter the contralateral orbital involvement usually appears within from twenty-four to forty-eight hours. Eagleton [2] described the thrombosis of the cavernous sinus resulting from aural infection as developing in a slightly different manner. Etiologically, it may retrograde; extending through the petrosal sinus to the cavernous sinus, or it may attack the cavernous sinus directly from diffuse suppuration of the bone marrow within the petrous tip of the temporal bone. In cases of this condition the exophthalmos is often slight and may be transient. These two points are important in considering a case of otitis media in which there are such mild and fleeting symptoms and signs. The changes in the fundus are: paleness of the nerve head, with occasionally some edema of the papilla; contraction or obliteration of the arteries, and normal or engorged veins (even thrombosed veins), and these are combined with impairment of vision, which is often marked. The conjunctival edema is at times pronounced, though it seems that the swelling of the lids is rather less than that seen in cases of orbital phlegmon. The early bilaterality is also of diagnostic value. A positive blood culture is conclusive as to the site of the thrombosis. This condition in the two cases cited is typical of the extremes to which this process may go. The latter of the 2 patients was dead in forty-eight hours. The former lived for more than four weeks. In this patient the bilateral exophthalmos had started to recede; blood cultures were negative, and the patient was free from signs or symptoms indicating meningitis. Signs of bronchopneumonia appeared, and the patient died of extensive pulmonary abscesses.

Exophthalmos is occasionally seen associated with an erysipelatous infection. Eagleton expressed the belief that those cases of proptosis in which erysipelas subsequently develops are primarily cases of phlebitic phlegmon in which the streptococcus causing the phlegmon has been transmuted into that of erysipelas, for it is well recognized that streptococci from an erythematous area of the skin in a person with scarlet fever may be the cause of phlegmonic cellulitis or may even cause osteomyelitis. The reverse of this process may often occur. A case in a child of 5 years of age illustrates this well. The patient was seriously ill with erysipelas for several days, when exophthalmos of the right orbit suddenly developed, which was followed in twenty-four hours by a similar condition of the left orbit. The first and natural conclusion was that thrombosis of the cavernous sinus had occurred. Antistreptococcus vaccine was administered, and the patient made a fairly rapid and uneventful recovery. The exophthalmos present, however, receded more slowly than did the other general symptoms. Some edema of the papillae of the optic nerves was present early in the course of this complication. It is rather likely that orbital thrombophlebitis, rather than frank phlegmon, was the immediate cause.

Tenonitis has been described by Shoemaker [11] as a cause for unilateral exophthalmos. It may be a part of orbital cellulitis or separate from this. In such instances it may be prodromal to panophthalmitis, and as such it is to be seriously considered. Suppurative tenonitis has been seen following surgical intervention on muscles, resulting in the loss of the eyeball. The symptoms of proptosis, chemosis, pain on motion, and restricted motion are present. Zentmayer [12] reported a case of tenonitis complicating typhoid fever. Shoemaker [11] stated that tenonitis has occurred after diphtheria and influenza. Idiopathic tenonitis is allied. it seems, to rheumatism and to gout. As he (Shoemaker) stated. "These diseases are often responsible for episcleritis, with which certain cases of tenonitis may be confounded. Ordinary care in the examination should make the differential diagnosis clear."

Syphilitic periostitis and tuberculous periostitis both cause exophthalmos of moderate degree. Periostitis and osteomyelitis caused by other organisms also occur, though uncommonly. The position of the diseased bone, the roentgen findings, the serologic reactions and other allied signs will be sufficient for a correct diagnosis. The chronicity of syphilitic periostitis and tuberculous periostitis is, perhaps, their outstanding feature. One case is illustrative. A young man had exophthalmos associated with a chronic pathologic condition of the lacrimal

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11. Shoemaker, W. T.: Disease of the Orbit, in Ball, J. M.: Modern Ophthalmology, ed. 3, Philadelphia, F. A. Davis Company, 1913.

12. Zentmayer, quoted by Shoemaker. [11]

sac. Dacryorhinocystotomy revealed tuberculous infection of the lacrimal sac, tuberculous osteomyelitis and formation of a sequestrum in the medial wall of the orbit. Recovery was ultimately complete, but the patient was under rather active observation for almost a year. Careful roentgen studies and injections of iodized poppy-seed oil 40 per cent into the lacrimal sac should have been done prior to any surgical intervention.

Engelking [13] described exophthalmos resulting from hyperplastic tuberculous lymph nodules, which he called tuberculoma, and expressed the belief that it probably originated from a condition of the conjunctiva. Exophthalmos of this type is, however, usually bilateral, though unequal in degree. Engelking stated that the picture was similar to that seen in pseudotumor of the orbit. In the case that he described the condition demonstrated a relationship to true lymphomatosis, a still closer relationship to inflammatory pseudotumor of the orbit and, finally, a relationship to the tuberculous processes and to other conditions resembling those which are grouped under the Mikulicz symptom complex. Relative to the diagnosis of periostitis and tenonitis, Shoemaker [11] said:

As both have many common symptoms . . . the differential diagnosis is generally not difficult. The boring deep seated pain, with nocturnal exacerbations, together with external signs, serve to implicate the periosteum in the inflammatory process. Tapping with the finger around the orbital margin, so as not to disturb the orbital contents, will serve often to locate the affection in the bones. If this can be done without pain, the bone or the periosteum is not involved. Vice versa, if gently pushing back the eyeball, with just sufficient force to reach the soft parts only, caused marked pain, we probably have to do with tenonitis or cellulitis. Tenonitis seems to cause the greater pain of the two. Syphilitic periostitis is generally a tertiary manifestation, but we should remember that it may, also, be found in the complex of the secondaries.

The paralysis of the third nerve involving the globe is likely to be periosteal in origin when the levator muscle continues to function. Syphilitic periostitis is more common in the roof of the orbit (the suppurative forms resulting from disease of the frontal sinus being excluded) and at the outer upper margin and rim of the orbit. The tuberculous form of periostitis selects other positions in the greater number of cases.

In Dunbar Roy's [14] case of orbital tuberculosis the condition was quite different. There was intense protrusion of the entire orbital contents; the bulbar conjunctiva was edematous and thickened, and palpation revealed a mass which seemed to fill the entire orbit, which was tender, and generally firm, but with a suggestion of fluctuation present.

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13. Engelking, E.: Ueber symmetrische Tuberkulome der Orbita, Klin. Monatsbl. f. Augenh. **70**:100 (March 10) 1923.

14. Roy, Dunbar: Tuberculoma of the Orbital Cavity, Arch. Ophth. **52**:147 (March) 1923.

Vision was destroyed. There was no glandular involvement about the body, and no history of tuberculosis in the family existed. Exenteration of the orbit confirmed the diagnosis.

In Rogers'[15] case the condition was characteristic of the syphilitic form of periostitis. The patient was a 60 year old man. There were exophthalmos, the classic pains, thickening of the orbital walls, absence of inflammatory changes, and accompanying uveitis. The Wassermann reaction was positive. Antisyphilitic treatment resulted in a negative serologic reaction and recession of all the symptoms and signs except proptosis of 5 mm., which was due to a permanent deposit of bone characteristic of syphilitic periostitis.

Pseudotumor is a term occasionally used for slowly developing exophthalmos caused by a low grade nonmycotic infection due probably to an unidentified organism. Shoemaker[11] reported a case of such a condition in detail and gave recommendations for treatment. The case was carefully studied also by de Schweinitz. Shoemaker stated: "In spite of invitations to exenterate the orbit, to enter the orbit, and to enter the antrum, all of which had academic merits but promised nothing beneficial to the patient, I continued with conservative and supportive treatment." De Schweinitz felt that the true nature of this lesion remained a matter of opinion, but the practical results were satisfactory.

Fry recently presented such a case before the Pennsylvania State Medical Association. The report of the pathologic examination of the enucleated eye is as follows:

The section shows old hyaline and fibrous tissue in abundance and some recently formed blood vessels. These are foci of lymphoid tissue well surrounded by connective tissue. There seems to be no acute process going on. This is not an actively malignant growth. It suggests chronic granulation tissue. The lymphoid inclusions suggest the possibility of Mikulicz' disease but are much smaller than in the one acceptably diagnosed case seen by the pathologist.

Figure 11 is the photomicrograph of a section of the diseased tissue of the growth in Fry's case.

Exophthalmos has been seen, as Foster[16] stated, after a penetrating wound of the orbit, associated with stiffness of the neck, convulsions, facial palsy and spasms of the throat, due, as one would surmise, to tetanus.

Acute exophthalmos may be symptomatic of an abscess in or about the lacrimal gland or may be due to acute dacryo-adenitis resulting from invasion by pyogenic organisms.

15. Rogers, R. M.: Periostitis of the Orbit. Am. J. Ophth. **7**:782 (Oct.) 1924.

16. Foster, Matthias: Diagnosis from Ocular Symptoms, New York, Rebman Co., 1917.

Exophthalmos resulting from chronic sinus disease is not common. Dowman's [17] case and St. Clair Thompson and M'Ilraith's [18] case of mucocele of the frontal sinus, Ferreri's [19] case of pulsating mucopneumatocele of the frontal sinus, Reverchon and Worms' [20] and Bower's [21] cases of mucocele of the ethmoid sinuses, Hajek's [22] case of mucocele of both the frontal sinus and the ethmoid sinuses and Peter's [23] case of exostosis or chronic periostitis of the ethmoid sinuses are all inclusive and classic examples of these somewhat rare complications. Figure 12 is a recent roentgenogram showing a mucocele of the ethmoid sinus and illustrates well a statement made previously in this paper to the effect that roentgen diagnosis plus careful clinical investigation by a competent rhinologist is necessary but adequate in cases of unilateral exophthalmos.
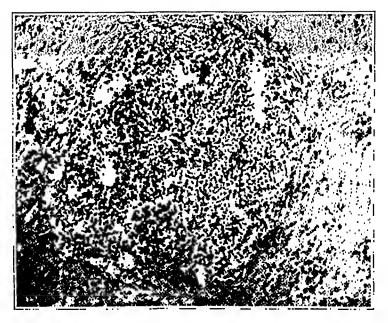
Fig. 11.—Photomicrograph of a section of the growth in Fry's case of pseudotumor of the orbit.

17. Dowman, C. E.: Giant Mucocele of the Frontal Sinus, J. A. M. A. **81:** 1014 (Sept. 22) 1923.

18. Thomson, St. Clair, and M'Ilraith, C. H.: Mucocele of the Frontal Sinus, J. Laryng. & Otol. **38:**365 (July) 1923.

19. Ferreri, G.: Muco-pneumatocèle "géant" bilatéral des sinus frontaux, Arch. internat. de laryng. **28:**973 (Sept.-Oct.) 1922.

20. Reverchon, L., and Worms, G.: Mucocèle fronto-ethmoidal, Arch. internat. de laryng. **28:**682 (June) 1922.

21. Bower, R. L.: Mucocele of the Ethmoid, Am. J. Ophth. **7:**218 (March) 1924.

22. Hajek, M.: Mucocele der rechten Stirnhöhle und des rechten Siebbeinlabyrinthes, Monatschr. f. Ohrenh. **58:**389 (May) 1924.

23. Peter, L. C.: Presentation of a case before the staff of the Graduate Hospital of the University of Pennsylvania, Oct. 12, 1932.

Cholesteatoma has been reported of such an extensive size as to break through the roof of the orbit and cause proptosis. Wolff [24] reported a study of the 18 cases which up to that time had been described in the literature. The growth originates from the diploe of the frontal bone as a result of chronic inflammation in the sinus. The earliest symptom is pain due to pressure, to be followed later by exophthalmos, swelling of the lids and choked disk. Wolff stated that roentgen diagnosis would be difficult except for the fact that there may be destruction of bone and the formation of fistulas. The frequent description of this growth, cholesteatoma, as a neoplasm is incorrect, as Knapp stated, and pathologists seem to be abandoning the belief that it is a form of tumor, i. e., neoplastic in origin.

Actinomycosis, anthrax, trichinosis, hyatid cyst and mycotic pseudotumor have been repeatedly reported, though not commonly, as the cause of unilateral exophthalmos. The diagnosis in the first of the conditions

Fig. 12.—Roentgenogram showing a mucocele of the ethmoid sinuses in a patient with unilateral exophthalmos.

mentioned should be the simplest, because of the general symptoms, as well as the local symptoms of abscess formation, the history of the patient and the laboratory studies. Studies of the cytologic features of the blood, and the orbital pains, plus a history of the characteristic muscular pains, will assist in cases of the third-mentioned condition. In the two last-mentioned conditions, however, the diagnosis depends largely on resection of the orbital wall, plus, perhaps, the elimination of every other possibility in any case under investigation. In cases of infection caused by Streptothrix the diagnosis is made on the basis of the symptoms of subacute or acute infection, suppuration, the occupation of the patient, perhaps, and the microscopic and macroscopic observations. The diagnosis of anthrax is based wholly on the acute suppurative process, the occupation of the patient (this being especially

24. Wolff, H.: Ueber ein Cholesteatom der Stirnhöhle, Beitr. z. klin. Chir. **130**:215, 1923.

important) and the gross and microscopic laboratory observations. Shipman presented a case of this condition before the College of Physicians of Philadelphia, in which the classic signs and symptoms of anthrax were present. In Pascheff's [25] case of actinomycotic tumor of the orbit the signs and symptoms were equally characteristic. The patient had unilateral exophthalmos, with ptosis of recent duration, and a history of orbital injury nineteen years before. Several abscesses, with fistulas which appeared six years later, led from the skin of the forehead in the neighborhood of the orbital rim. The cultures showed Streptothrix. These fistulas healed after several months of medication with potassium iodide. Two years later the condition recurred and was most severe, leading to complete immobility of the proptosed eyeball. This time the tumors were removed by surgical intervention, with complete and rapid cure. These mycotic pseudotumors of the orbit may continue without fistulas for several years. In this form they are not extremely rare. González' [26] patient presented paralysis of the third nerve, exophthalmos, mydriasis and paralysis of accommodation, cutaneous and corneal anesthesia and corneal ulceration with hypopyon. If the correct diagnosis is not made, the condition will continue to complete immobility of the eyeball, blindness from atrophy of the optic nerve and emaciation of the patient, with the development of multiple fistulas as the terminal stage.

Rather recently, O'Brien and Leinfelder [27] reported a case of a somewhat different condition, in which surgical exploration was without success, and death occurred from thrombosis of a cavernous sinus. In this case cultures positive for Streptothrix were obtained from the brain at the necropsy. Matthias Foster [16] discussed the symptomatology in a case of trichinosis of the ocular muscles, which he diagnosed largely on the basis of oculomotor paralysis of recent duration which could not be ascribed to any lesion of the afferent nerves, the presence of a doughy edema of the lower lid and other general symptoms of this infection. Hydatid cyst is due to the echinococcus, and cystic degeneration may be due to Cysticercus cellulosae. Both the conditions just mentioned are rare, especially the second. Berlin, Parsons, Aniceto-Solares,[28] Lagrange, L'Heureux and Wood [29] have written about these unusual

25. Pascheff, C.: Ueber eine Streptothrix-Strahlenpilz-Geschwulst der Orbita, Ztschr. f. Augenh. **47**:109, 1922.

26. González, J. de J.: Tumor micósico de la órbita, Rev. cubana de oftal. **4**:76, 1922.

27. O'Brien, C. S., and Leinfelder, P. J.: Unilateral Exophthalmos, Am. J. Ophth. **18**:123 (Feb.) 1935.

28. Aniceto-Solares: Les kystes hydatiques de l'orbite, Arch. d'opht. **39**:406 and 491, 1921.

29. Wood, D. J.: Hydatid Cysts of the Orbit, Brit. J. Ophth. **9**:4 (Jan.) 1925.

conditions. Wood was so fortunate as to observe 3 cases, which he described. He stated that the diagnosis is uncertain, apart from positive results of examination of material removed by aspiration, but the negative Wassermann reaction, the absence of inflammation, the frequent attacks of blindness without the presence of a pathologic condition of the fundus, and a palpable tumor are all suggestive, especially if the patient lives or has lived in a land where hydatid disease occurs. According to Wood's experience, these cysts may lie either free in the orbit or within the muscle zone.

The last of the conditions to be considered in this section of inflammatory causes is xanthomatosis of the orbit allied to the Schüller-Christian syndrome—a syndrome of exophthalmos associated with diabetes insipidus and with large defects in the bones of the skull (Wheeler[30]). The Schüller-Christian syndrome occurs essentially bilaterally, but because of the allied xanthomatosis, which follows, it seems proper to mention it here. The condition is similar to cholesteatoma in the indefiniteness of its origin. Knapp,[31] Heath,[32] Pincus[33] and others have written relative to this disease. Xanthomatosis of the orbit, a part of the syndrome just mentioned, has been described by Knapp[31] as a unilateral, tumorous, hyperplastic condition of the reticulo-endothelial cells, resulting from the infiltration of lipoids in excess of the body fluids. Unilateral exophthalmos, accompanying neuritis of the optic nerve, rarefaction of the roof of the orbit and the presence of a palpable mass in the orbit, apparently continuous with the frontal bone, were the characteristic features in Knapp's case. Evisceration of the orbit is alone confirmatory of the diagnosis.

DISEASES OF THE BLOOD, THE LYMPH AND THE HEMATO-POIETIC SYSTEMS

Diseases of the blood, the lymph and the hematopoietic system are responsible for many cases of unilateral and bilateral exophthalmos. Rickets, scurvy and hemophilia are the first to be mentioned. These conditions result in subperiosteal hemorrhages, deep in the orbit, which occur abruptly and are accompanied by hemorrhagic extravasations into the skin of the upper and the lower lids, especially the lower lids.

30. Wheeler, J. M.: Schüller-Christian Disease (Xanthomatosis), Arch. Ophth. **11**:214, (Jan.) 1934.

31. Knapp, A.: Xanthomatosis of the Orbit, Arch. Ophth. **11**:141 (Jan.) 1934.

32. Heath, P.: Ocular Lipoid Histocytosis and Allied Storage Phenomena, Arch. Ophth. **10**:342 (Sept.) 1933.

33. Pincus, F.: Ueber „Cholesteatom" der Orbita, Klin. Monatsbl. f. Augenh. **90**:145 (Feb.) 1933.

Otherwise, the general symptoms which identify these conditions should be sufficient for making a correct diagnosis.

In regard to these conditions, some rather important dictates relative to unilateral exophthalmos should be emphasized. The condition must be investigated immediately, certainly before any surgical intervention is considered, by the following: (*a*) roentgenograms taken early; (*b*) studies of the basal metabolic rate; (*c*) the Wassermann test; (*d*) studies of the nose and throat; (*e*) careful studies of the blood and of the cytologic features of the blood.

Lymphosarcoma (lymphoblastic), lympho-endothelioma, acute and chronic leukemia and, possibly, Mickulicz' disease are allied conditions. With the exception of the leukemias, the blood picture is not especially characteristic. Leinfelder and O'Brien [34] stated: "Characteristically, all types of lymphoma show progressive, painless enlargement of the cervical lymph nodes, increase in the size of the spleen and liver, and in the later stages, secondary anemia and cachexia." These are common. With the exception of the signs, the diagnosis must be based, usually, on observations at postoperative or postmortem microscopic examination. In cases of lymphosarcoma, the lids are usually involved by nodules; the exophthalmos is apparently of moderate degree, and age seems to have no relationship to the condition. Leinfelder and O'Brien [34] reported 3 cases of lympho-endothelioma, in 2 of which the patient had unilateral exophthalmos. They stated the belief that lympho-endothelioma is a rare condition, similar in the general symptomatology to Hodgkin's disease. In 2 of their patients cranial extensions developed, and a third patient presented a Horner's syndrome resulting from enlargement of the cervical lymph nodes. The diagnosis depended wholly on the microscopic picture of the tumor. Figure 13 illustrates one of their cases of unilateral exophthalmos associated with this malignant lymphoma of endothelial nature.

Acute and chronic leukemia are not commonly accompanied by exophthalmos. In 96 cases of lymphatic leukemia reviewed by Reese and Guy,[35] 2 per cent of the patients showed exophthalmos. The blood picture in this condition is characteristic. One of Reese and Guy's patients had a count of 160,000 white cells per cubic centimeter, 94 per cent of them being small lymphocytes. In cases of this condition retinal hemorrhages are common; the retinal vessels have a pale, even milky, appearance, and there is often a soft edematous swelling of the lids. Figure 14 illustrates one of O'Brien's cases of this condition.

34. Leinfelder, P. J., and O'Brien, C. S.: Lymphoma of the Eye and Adnexa, Arch. Ophth. **14**:183 (Aug.) 1935.

35. Reese, A. B., and Guy, L.: Exophthalmos in Leukemia, Am. J. Ophth. **16**:718 (Aug.) 1933.

Recently, a case of Adler's, from the Wills Hospital, illustrated certain difficulties in the diagnosis of this condition. The case was one of unilateral exophthalmos in a woman aged 65. The fundus showed no characteristic pathologic changes. The white cells of the blood numbered 20,000 per cubic centimeter, with 28 per cent polymorphonuclears and 68 per cent large adult and immature lymphocytes. Two pathologists considered the case to be one of acute lymphoblastic leukemia, while two others called it one of acute infectious mononucleosis. The cause for the confusion is not unreasonable, but the patient was not definitely benefited thereby.

Cases of Mickulicz' disease, it seems, may belong to the cases of the leukemias. This condition consists of chronic bilateral enlargement

Fig. 13.—Unilateral exophthalmos caused by malignant lymphoma (O'Brien and Leinfelder).

Fig. 14.—Unilateral exophthalmos associated with acute lymphatic leukemia (O'Brien).

of the lacrimal and the salivary glands in which the characteristic microscopic observation is marked infiltration by lymphocytes, and also by endothelioid and giant cells, which are occasionally present. Wolff [36] stated that "the etiology is much disputed and probably varies in different cases."

Exophthalmos has been seen in association with Hodgkin's disease, though, considering the relative frequency of the latter condition, associated proptosis is rare. A case of Hodgkin's disease was observed at the Walter Reed Hospital, in which, after cachexia had advanced rather far, unilateral exophthalmos developed. The eye in which it occurred

36. Wolff, E.: Pathology of the Eye, Philadelphia, P. Blakiston's Son & Co., 1935.

was on the opposite side of the body to that in which there was the greatest amount of enlargement of the cervical lymph nodes. The possibility of a Horner's syndrome contralaterally was eliminated by the finding of a palpable mass in the outer, superficial, angle of the orbit. Leinfelder's and O'Brien's [34] patient had bilateral exophthalmos, with a nodule adherent or attached to each external rectus muscle. It is likely that this condition was similar to that I observed. The microscopic picture of any one of the enlarged lymph nodes is classic, though Hodgkin's disease has been misdiagnosed at various times as tuberculous adenitis.

Myeloblastoma and myelogenous leukemia are similar to the leukemic conditions except for the presence of premyelocytes and myelocytes in the blood stream. The white cells usually number over 100,000; progress is rapid, and massive hemorrhages occur into the tissues of the
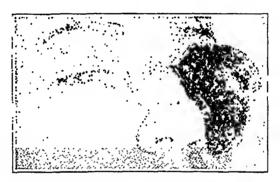
Fig. 15.—Unilateral exophthalmos associated with myelogenous leukemia (Reese and Guy).

orbit. The blood picture cannot be mistaken. The patient whose case was reported by Reese and Guy [35] had 510,000 white cells per cubic centimeter, Burchell's report in their case under the heading "differential count" being "Myeloid series over 90 per cent and morphologically that of an extremely embryonal type of cell . . . About 5 per cent seem to be differentiating into the eosinophile type." Figure 15 illustrates one of their cases. The massive late hemorrhages are rather well seen. These tumors, when green and infiltrating, are spoken of as chloromas. The condition in Barnert's [37] case was characteristic. In this case the disease advanced into the bones of the skull and proved fatal in one month. In regard to tumors, Piney [38] stated: "The mode of infiltration of the tissue does not seem to differ from that characteristic of malignant tumors in general." The blood picture in his case consisted of 85 per

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37. Barnert, C.: Report of a Case of Chloroma of the Orbit, Arch. Ophth. **53**:454 (Sept.) 1924.

38. Piney, A.: Diseases of the Blood, Philadelphia, P. Blakiston's Son & Co., 1928.

cent myeloblasts and 15 per cent premyelocytes. Figure 16 illustrates a case of O'Brien's showing an orbital tumor. The patient had a normal blood picture, but the microscopic diagnosis was that of an undifferentiated tumor of the hematopoietic system, and roentgenograms showed a small area of rarefaction in the medulla of one tibia.

SPACE-TAKING ORBITAL LESIONS

Relative to the space-taking lesions which appear in the orbit, there are five general classes to be considered. The first is true cyst; the second, gumma; the third, a benign neoplasm; the fourth, a malignant neoplasm which is primary in the orbit or in the eyeball and which, by extension through the globe, invades the orbit, and the last, an orbital metastatic neoplasm secondary to a tumor of the nasal accessory sinuses, the liver, the kidney, the adrenal glands or some other organ. All these space-taking lesions have as a common symptom slowly developing exophthalmos, usually with but few if any inflammatory changes. Changes in the fundus are rare save in those patients with primary

Fig. 16.—Myeloblastoma of the orbit (O'Brien).

retinal, choroidal or papillary involvement, and the age limits are roughly individual and peculiar to certain conditions. Less common signs and symptoms are: duration of the exophthalmos, roentgen changes in the bones of the orbit and changes in the normal density of the orbital contents, various oculomotor disturbances, the direction of the proptosis, inflammatory signs and the presence of a primary neoplasm in some extra-orbital portion of the body.

Pseudotumor is again mentioned here, for though it is inflammatory in origin and neither cystic nor neoplastic, its inclusion herein is a logical addition, by reason of the name by which it is known. (See the section on inflammatory conditions of the orbit.)

The first class will include dermoid cyst and sebaceous (retention) cyst. One of the classic examples of the latter is the case of an 18 year old girl with exophthalmos which started at the age of 7 years. In the previous two years of the patient's life a mass could be palpated constantly in the orbit. There was no oculomotor or visual disturbance, but diplopia had developed recently. A Krönlein resection of the orbital wall showed an orbital cyst containing rolls of hair and much sebaceous material, the internal wall of the cyst being adherent to the sheath

of the optic nerve. Complete removal was accomplished save for that portion which was attached to the neural sheath of the optic nerve. The length of time the exophthalmos was present, its onset at an early age, its slow development and the absence of any other pathologic signs were sufficient for the diagnosis. A roentgenogram showed a mass in the orbit, in the retrobulbar space, but this was not of sufficient density to make one consider fibroma or osteoma. Lipoma might have been considered from the standpoint of the age of the patient, the development of the growth and its duration, but the noncompressibility of the palpable tumor, the direction of the proptosis (down and in) and the density of the roentgen shadow made cyst the more probable diagnosis. Following the surgical removal of this cyst, further investigation into the history (obtained from the patient's uncle, a physician) revealed a significant fact. Five years prior to the resection of the orbital wall, a rhinologist in the patient's place of residence had evacuated a cyst, which contained sebaceous material, from the ethmoid region of the same (left) side. This had ruptured spontaneously into the patient's middle nasal meatus. We were inclined to consider that cyst, which was incompletely removed at this time, as the same cyst which was removed subsequently.

An interesting oil cyst, occurring in the retrobulbar and subperiosteal region, was described in 1923 by Knapp,[39] with a complete pathologic report, the contents being of such a material that one might believe the growth belonged to the group of dermoid tumor. According to Knapp and, even earlier, to Lagrange,[40] this type of cyst has a predilection for the prelacrimal region within the orbit.

A similar cyst was seen recently, in Baer's service at the Wills Hospital. The patient was operated on by Rankin. The contents of the sac were identical to those found in the cyst described by Knapp, and the cyst was in an identical location. In fact, in this case the pedicle of the cyst was adherent to the prelacrimal periosteum, and the cyst had eroded a marked depression into the external surface of the supraorbital region of the frontal prominence.

Gumma of the orbit is apparently becoming much more rare than formerly. Kemp,[41] in discussing syphilis of the orbit, gave a detailed description of 4 cases of this condition. All the patients had acquired syphilis some years previously; all had pain in the affected eye, which was worse at night, and unilateral exophthalmos, with paralysis of some of the extra-ocular muscles; some even had complete ophthalmoplegia externa, and all showed abnormal pupillary reactions on the affected

39. Knapp, A.: Oil Cyst of the Orbit, Arch. Ophth. **52**:163 (March) 1923.
40. Lagrange, quoted by Knapp.[39]
41. Kemp, J. E.: Syphilis of the Orbit, Arch. Dermat. & Syph. **8**:165 (Aug.) 1923.

side. Three of the patients showed fundic pathologic changes of the nerve head. Kemp expressed the belief that the pain was the most characteristic symptom in cases of this condition. In addition to those cases of unilateral exophthalmos in which there is a true gumma, in other cases also syphilis may be the cause of exophthalmos—with many of the other symptoms just described due to periostitis and, or with, osteitis. In one case, which was observed in Peter's service, at the Graduate Hospital, the condition was characteristic. The patient was a 22 year old colored girl, with a positive Wassermann reaction, with the characteristic pains described by Kemp and with exophthalmos of 1 cm. in the right eye, accompanied by complete paralysis of the third nerve. Each fundus showed a pathologic condition of the blood vessels unusual

Fig. 17.—Fibroma of the orbit (Benedict's case). A, appearance of the patient before removal; B, appearance of the patient after removal; C, the tumor.

for a person of the patient's age. Recovery, with the patient under antisyphilitic treatment continued for three months, was uneventful.

Of the benign tumors reported as a cause of unilateral exophthalmos, fibroma, lipoma, osteoma and osteo-fibroma, and neurofibroma will be discussed first. Fibroma seems to be made up of white connective tissue, showing some hyaline degeneration, and mitotic figures are extremely rare. It may recur, however, if not completely removed. The condition in the case of Stokes and Bowers [42] is typical of this rather uncommon tumor. Benedict's case [43] (fig. 17 A, B and C) is detailed here. The patient, 34 years of age, had exophthalmos of 14 mm. in the left eye, due to a tumor, without diplopia and with a normal

42. Stokes, W. H., and Bowers, W. F.: Pure Fibroma of the Orbit, Arch. Ophth. **11**:279 (Feb.) 1934.

43. Benedict, W. L.: Personal communication to the author, September 1936.

fundus. Removal of the tumor was achieved through an external, lateral periorbital incision. The tumor was 2.5 by 2 by 3 cm. in size, encapsulated, slightly vascular, and pigmented on the anterior end. There were no large vessels and no firm attachments to the orbit.

Relative to osteoma, it is at times difficult to decide whether it is due to hyperplasia or heteroplasia of bone. Certainly, the exostoses one sees in chronic disease of the ethmoid sinuses are of the latter type— due to chronic inflammation or to chronic traumatic insult. On the other hand, true osteoma due to hyperplasia of bone appears in the orbit in situations where it is unlikely that the tumor can be due to preexisting inflammation. The roentgen picture shown in figure 18 is typical of that of early but definite osteoma. This heteroplastic form of bone formation is well illustrated by the case of pseudo-osteoma reported by Van Duyse and Moret.[44] The patient had marked exophthalmos, which was corrected surgically by the removal of two different tumors, one in the retrobulbar space and the second in the temporal fossa. The
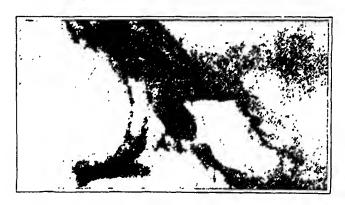
Fig. 18.—Roentgenogram showing osteoma of the orbit.

diagnosis of the orbital mass, based on microscopic examination, was calcified fibrolipo-epithelioma, and that of the mass in the temporal portion of the cranium was calcified epithelio-osteoma.

Neurofibroma, or fibroma molluscum, of the plexiform variety, appears rather commonly on the face and in the orbit. The tumor is not neurogenic but is derived from connective tissue, probably from the sheath of Schwann. Figure 19 shows a typical neurofibroma of the orbit. Not only do patients with this condition have unilateral exophthalmos resulting from the retrobulbar growth of the new tissue, but the tumor in many instances causes a deformity of the floor and roof of the orbit. This was so in each of the 4 different cases observed by me. The possibility that fibroma may become sarcomatous later is illustrated well by Zentmayer's[45] case of osteosarcoma. His patient had

44. Van Duyse, D., and Moret: Pseudo-ostéome de l'orbite, Arch. d'opht. **41**:129 (March) 1924.

45. Zentmayer, W.: Osteosarcoma of the Orbit, with Unusual Surgical Complications, Am. J. Ophth. **9**:736 (Oct.) 1926.

an indefinite history of mild trauma six years earlier. The exophthalmos developed slowly for four years, then abruptly began to advance rapidly. The true diagnosis of the type of tissue present had to be made microscopically.

Myxoma and chondromyxoma have both been reported as the cause of unilateral exophthalmos. The first of these consists of cells of an embryonal type, grows very slowly and occasionally becomes sarcomatous. The chondroma has typical fetal cartilage cells and, occasionally, cartilage with stellate cells. It is at times myxomatous as well. This tumor is encapsulated, hard and nodular. It seems that when the encapsulation becomes imperfect the growth spreads rapidly, though it does not metastasize. Ewing [46] reported a case of this condition in detail. Klionsky [47] reported a case of myxoma of the orbit and of the frontal sinus of such a size that enucleation of the eye was necessary to remove the entire mass. Considerable destruction of bone was present in the roof of the orbit.

Fig. 19.—Neurofibroma of the orbit.

Cylindroma was at one time thought to be endothelial. Now it is more commonly considered as epithelial. The cells are arranged in cylindric spaces which are filled with hyalin. This type of tumor occurs most commonly as the ordinary mixed cell tumor of the parotid gland. Zentmayer [15] cited a case of this variety as connected with the lacrimal gland, and Agnes Baron [48] reported a case of this condition, giving in great detail the clinical and microscopic features. Her observations permitted her to consider the lacrimal gland as an altered salivary gland and to designate this type of tumor as prognoblastoma.

Psammoma is truly endothelial, consisting of whorls of endothelial cells with areas of centrally disposed laminated calcification. Karsner [49]

46. Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928.

47. Klionsky, G.: Displacement of One Eyeball Due to Myxoma of the Frontal Sinus, Lancet 1:126 (Jan. 17) 1925.

48 Baron, Agnes: Zur Stellung der Orbitalcylindrome im onkologischen System als Progonoblastome, Arch. f. Ophth. 113:31, 1924.

49. Karsner, H. T.: Human Pathology, Philadelphia, J. B. Lippincott Company, 1926.

stated that mitotic figures are not uncommon. This condition is rare and seems to be connected with chronic nasal disease. In the case described by von Eicken [50] the patient showed roentgenographic signs of disease of the ethmoid sinuses, with marked shading of the ethmoid and frontal sinuses. The exophthalmos had been developing over a period of six years. Eicken reported the findings at operation as follows:

Large masses of the tumor tissue were removed, and it was discovered that all the dividing walls of the ethmoid bone and the anterior wall of the sphenoid sinus were lacking. The tumor mass crunched under the spoonlike sand. Extensive hemorrhage prevented a reliable radical extirpation. Sixteen days later, following cleansing and cocainization of the nasal cavity, examination revealed a firm tumor, which nearly filled the cavity, in the depths of the inferior nasal passage. Operation demonstrated this tumor to be a firm clublike swollen structure containing granulations and vestiges of tumor. Healing occurred in eleven days.

Adamantinoma is a cystic tumor which arises from the paradental epithelial débris. Sooner or later the capsule of this tumor ruptures, and the disease extends rapidly into the antrum, the orbit and the cranial cavity. Ewing [46] reported 2 instances of such a condition. In figure 20, *A* and *B* are a photograph and a roentgenogram, respectively, illustrating O'Brien's case of adamantinoma of the orbit.

Lymphangioma and hemangioma are rather uncommonly the cause of unilateral exophthalmos. They consist of a tangled network of dilated blood vessels or lymph vessels. They are usually congenital, tend to increase in size and may become malignant through sarcomatous changes. Hemangioma is usually bluish and is, perhaps, the more common of the two. In the case reported by Franklin and Cordes [51] the tumor is typical of lymphangioma. Marked proptosis was present. Exploratory operation through the conjunctival cul-de-sac showed a cyst with clear watery contents, which was removed by resection of the orbital wall three months later. The proptosis had started in the early months of the child's life. The mass could be palpated in the orbit, was not tender, was freely movable and fluctuated somewhat. Microscopic examination of the tissue was necessary for a correct diagnosis. Ring [52] and Jones [53] each reported interesting cases of orbital heman-

50. von Eicken, C.: Ueber ein Psammom des Siebbeins und der Keilbeinhöhle, Schweiz. med. Wchnschr. **52**:495 (May 25) 1922.

51. Franklin, W. S., and Cordes, F. C.: A Case of Orbital Lymphangioma, J. A. M. A. **83**:1741 (Nov. 29) 1924.

52. Ring, G. O.: Orbital Cavernous Hemiangioma with Pulsating Exophthalmos, Am. J. Ophth. **7**:946 (Dec.) 1924.

53. Jones, A. C.: Orbital Hemangioma Treated with Radium, Northwest Med. **24**:11 (Jan.) 1925.

gioma. In Ring's case the tumor was accompanied by pulsating exophthalmos and was the result of extension of a vascular growth into the orbit from the face, which was first noticed there when the patient was 2 years of age. The patient had had a ligation of the common carotid artery six years before. This failed to stop the progress of the intraorbital extension. Jones' patient had a blue prominence, which first appeared in the left upper lid at the age of 2 weeks. Within six years it had progressed appreciably, and was accompanied with moderate exophthalmos, vertical displacement of the eyeball and diplopia; there was no bruit, and it did not change in size with any change in the position of the patient's head.

Plasmoma, or tumor of the extramedullary plasma cells, has been reported. Ewing [46] stated that such tumors are commonly classed as

Fig. 20.—*A*, photograph, and *B*, roentgenogram, showing adamantinoma of the orbit (O'Brien).

lymphosarcomas but, "being as a rule, benign processes with very indistinct neoplastic properties, it is highly important that they should be separated from the malignant lymphosarcomas. They may recur after extirpation and become associated with a chronic cachexia." This confusion in diagnosis was illustrated in the case of plasmoma of the orbit described by Przybylska.[54] The patient's first operation had been done in a small continental city, and the examination of the tissue removed at that time resulted in a diagnosis of sarcoma. Secondary operation showed that the tumor had invaded the entire orbit, the maxillary antrums and the ethmoid cells, and exenteration of the orbit was necessary. The patient died after eighteen months, with signs of general cachexia.

54. Przybylska, J.: Plasmome de l'orbite, Ann. d'ocul. **161**:198, 1924.

Endothelioma is more commonly known by Cushing's preferred term, meningioma. Ewing [46] stated: "These tumors while not encapsulated show no great invasion capacity, and do not metastasize." On the other hand, certain cases have been reported in which either extensions or metastases in the neighborhood of the original tumor appeared. The roentgenogram in my case is shown in figure 21. In cases of this tumor the diagnosis is not definitely certain until after operation. The adult age of the patient, the slowly developing exophthalmos, the absence of pain and of tenderness, the negative findings in the fundus, the absence of any history of trauma and the extensive roentgenologic findings are necessary factors in making a preoperative diagnosis in any case. Cohen and Scarff [55] reported a case of a similar condition in which surgical intervention was attempted, but without complete success. The microscopic examination was reported to show fibroblastic tumor tissue, appearing as whorls about the blood vessels, with psammoma bodies present and with clusters of endothelial cells apparently

Fig. 21.—Roentgenogram showing meningioma of the left orbit of a patient with exophthalmos.

normal and, in general, on the surface of the fibrous tissue. Owing to the orbital invasion in this condition, surgical intervention is not satisfactory, except that which would be extensive and disfiguring and, in addition, ineffective as regards complete removal of the neoplasm.

Other cases of endothelioma occurring in the retrobulbar space have been reported, but these tumors did not show the characteristic type of tissue. The 2 cases reported by Williamson-Noble [56] demonstrated this. Both his cases were in children. In the first case the tumor seemed to appear as the result of postoperative trauma in a case of squint, and microscopically (to this author) seemed to be a plasmoma rather than a meningioma. The growth invested the optic nerve closely and seemed to arise from the arachnoid about the nerve. The tumor in the second

55. Cohen, M., and Scarff, J. E.: Unilateral Exophthalmos Produced by Meningioma of Middle Cranial Fossa, Arch. Ophth. **13**:771 (May) 1935.

56. Williamson-Noble, F. A.: Endothelioma of Orbit, Brit. J. Ophth. **7**:222 (May) 1923.

case contained not only endothelial cells but also blood spaces, fibrous tissue, cartilage and bone. Williamson-Noble stated:

It is not conceivable that these cells would form cartilage and bone. The tumor, therefore, was not a typical endothelioma. Assuming that some pluripotential cells were cut off during embryonal life, later to resume activity, the growth could be a teratoma of the orbit—though careful search failed to show evidence of any other structures.

Rhabdomyoma has been reported as the cause of unilateral proptosis. This tumor originates in preexisting striated muscle, though it may occur in heterologous tissue. The tumor is rare and occurs usually in the tongue, heart muscle ("as the result of congenitally misplaced embryonic striated muscle"—Ewing) and skeletal muscle. It rarely metastasizes. Redslob [57] reported on the 3 cases described in the literature and included a fourth. All of them occurred in children between 2 to 5 years of age, and caused irreducible exophthalmos. In his case, in which there was a tumor on the external rectus muscle, the growth recurred. Microscopic examination of the second growth showed the tissue cells of typical round cell sarcoma, quite unlike the picture seen in the first mass removed. Redslob stated: "There can be no doubt that this second tumor is a recurrence, since the primary tumor showed zones of transition in which the morphologic degeneration of the tumoral rhabdocytes could already be plainly traced."

Tumors of neural or glial tissue, which may be extraneural as well as intraneural, and extra-ocular as well as intra-ocular, are diagnosed according to the type of the predominant cell. The work of Bailey, of Cushing, of Penfield and of others, has resulted in a differentiation of thirteen different types of glioma, depending on the resemblance of the cells to the embryonal cells at various ages; thus, medulloblasts, spongioblasts and neuroblasts are observed. Through stages of further differentiation the medulloblasts would produce oligodendroglioma; the spongioblasts, astrocytoma, and the neuroblasts, neuroma. Neuroblastoma shows, histologically, few, if any, glia fibers and is made up of cells resembling the primary neuro-ectoderm. It usually affects the adrenal glands in children and metastasizes frequently. Figure 22 *A* and *B*, from Lowenburg's pediatric service at the Mount Sinai Hospital of Philadelphia, shows the typical appearance in a case of this condition. The picture shown in *A* was taken three months before that shown in *B*, the latter having been taken a few days before the death of the child. *B* shows the huge extension at the midline of the frontal bone, which grew much more rapidly than did the metastatic growth in the retrobulbar space. Figure 23 shows a neuroblastoma reported by

57. Redslob, E.: Rhabdomyome de l'orbite à évolution atypique, Ann. d'ocul. **161**:721, 1924.

O'Brien, which was primary within the orbit and which occurred in an older patient.

Neuroma is essentially a benign glioma and is composed principally of both medullated and nonmedullated fibers, though a careful search may show some cells. It has been reported frequently as adherent to the optic nerve and can be diagnosed only at operation. The growth is very slow and is not accompanied by any characteristic symptoms. In Stieren's [58] case of neuroma of the orbit, the course and the observations
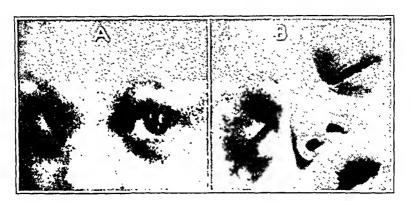
Fig. 22.—Appearance of the patient in Lowenburg's case of neuroblastoma (A) at the time of the earliest exophthalmos and (B) shortly before death. The metastatic growth in the forehead can be seen.

Fig. 23.—Appearance of the patient in O'Brien's case of neuroblastoma.

at microscopic examination were not quite typical of this condition. The patient was 28 years of age when the exophthalmos began to develop. It progressed slowly. Progressive hyperopia developed in the proptosed eye; few, if any, pathologic changes appeared in the fundus, and a roentgenogram showed a faint shadow in the retrobulbar space. Removal was uneventful and not difficult, and microscopic examination confirmed

58. Stieren, E.: Neurofibroma of the Orbit, Am. J. Ophth. 6:176 (March) 1923.

the diagnosis of a neural tumor. In Benedict's [59] case the condition was more typical, and the case was interesting in that this author was able to follow the patient, a 7 year old child, for many years after surgical intervention. In this patient exophthalmos of eight months' standing had advanced rapidly within a month, and at the time of operation there was unilateral exophthalmos of 8 mm. At the removal of the tumor, through a superolateral periorbital incision (without resection of the orbital wall), it was found to lie within the muscle cone, was 1 cm. in length, and at the bulbar attachment of the optic nerve, where the tumor arose, it was no thicker than the optic nerve. Figure 24 A, B and C illustrates this case.

Spongioblastoma polare is a different glioma, which occurs, as a rule, in the optic nerve, the optic chiasm or the optic tract. It consists principally of either bipolar or unipolar spongioblasts. In Kiehle's [60] recently reported case of this condition, the growth was typical of this type of

Fig. 24.—Appearance of Benedict's patient with glioma of the optic nerve (A) before removal of the growth, (B) after removal and (C) ten years later.

tumor. This tumor occurs in children or in very young adults and is accompanied with slowly developing exophthalmos, which at some time begins to increase suddenly. The patient shows optic neuritis, usually of the retrobulbar type; retinal hemorrhages are common, and blindness usually occurs at an early period in the progress of the disease. The certain diagnosis of the type of tissue present must be made microscopically.

Neuro-epithelioma is rare in the brain and in the spinal cord but is a common lesion in the retina. It consists of primitive spongioblasts which tend to form many rosettes. Retinoblastoma shows a lesser differentiation of the cells and is more malignant, and it is considered that the rosettes are made up of primitive rods and cones. Exophthalmos resulting from these tumors would indicate extension of the tumor from

59. Benedict, W. L.: Removal of Orbital Tumors, Surg., Gynec. & Obst. **58**:383 (Feb. 15) 1934.

60. Kiehle, F. A.: Tumor of the Optic Nerve, Arch. Ophth. **15**:686 (April) 1936.

the globe through the scleral cribriform plate into the orbit. Surgical intervention or, at least, radiotherapy should have been carried out in cases of these tumors long before this complication has occurred.

The syndrome of tumor of the sphenoid ridge, described by Groff and Alpers,[61] is not uncommon. It illustrates a form of meningioma of the greater wing of the sphenoid bone, intracranial in origin, and demanding extensive neural surgical intervention because of its certain progress. The outstanding signs of the syndrome, as outlined by Groff [61a] in his report of cases, are as follows: primary atrophy of the optic nerve; defects in the visual field, which usually take the form of complete homonymous hemianopia (though the defect may be quadrantic); paralysis of the third nerve; unilateral impairment of the olfactory nerve, and unilateral exophthalmos. Additional symptoms, such as monoplegia, impairment of memory and disturbances of the pituitary body, are observed. Figure 25 shows the characteristic roentgen findings in this condition.
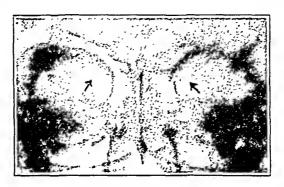
Fig. 25.—Roentgen findings in a case of tumor of the sphenoid ridge (Groff).

Sarcoma, mixed cell tumor and carcinoma have all been responsible for unilateral exophthalmos. These conditions are of grave prognostic import, however. Occasionally the patient is free from metastasis for many years, and other patients die early with involvement of the mediastinum and liver. Figure 26 shows the eye of a young colored man with primary retrobulbar sarcoma. Complete orbital exenteration was done, and the patient is still alive eighteen years later. Figure 27 shows the orbit, as it appears at present, of a patient with intra-ocular sarcoma with secondary orbital extension. This photograph was taken three years after a recurrence. The results of radium therapy are evident in the tissues. Figure 28 shows the appearance of a child, a patient of O'Brien's, with primary sarcoma. This case illustrates well the fact that sarcoma is more common in younger patients. Carcinoma occurs

61. (*a*) Groff, R. A.: Syndrome of Meningeal Fibroblastoma Arising from Lesser Wing of Sphenoid Bone, Arch. Ophth. **15**:163 (Feb.) 1936. (*b*) Alpers, B. J., and Groff, R. A.: Parasellar Tumors: Meningeal Fibroblastomas Arising from Sphenoid Ridge, Arch. Neurol. & Psychiat. **31**:713 (April) 1934.

more commonly in those of later years. In Howland's [62] case of mixed cell sarcoma in a girl 20 years of age, in which the tumor was removed by him from the orbital muscle cone by Krönlein resection, the condition was illustrative of that in such cases. The exophthalmos was of from four to six months' duration, there were no inflammatory reactions, the fundus showed no pathologic changes, and there was no definite paralysis of oculomotor nerves, but a solid mass, which was somewhat movable, could be palpated deep in the orbit. At operation it was found to be encapsulated and was removed rather readily. Further radiotherapy was carried out, after microscopic examination and
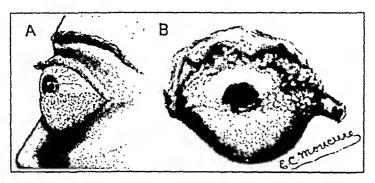
Fig. 26.—A, primary retrobulbar sarcoma; B, the eyeball after exenteration.
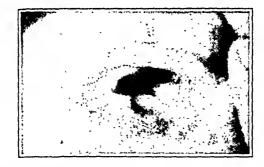
Fig. 27.—The socket of a patient with intra-ocular primary sarcoma three years after surgical intervention for removal of a recurrent orbital growth and subsequent radium therapy.

the receipt of the pathologist's report. The serious factor in cases of this condition is the early necessity for a diagnosis in the course of the condition: It should be determined that the condition of the orbit calls for surgical intervention and that it must be explored for more certain diagnosis. Gumma of the orbit might simulate this tumor, but other signs and symptoms are present in cases of the latter condition which are not seen in cases of a sarcomatous neoplasm.

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62. Howland, A.: Personal communication to the author, from the Wills Hospital, October 1936.

Both carcinoma and sarcoma have extended from the antrum, from the ethmoid sinuses and from the septum into the orbit. The complications are not uncommon and are often terminal. Psammoma has been reported as extending from the ethmoid sinuses; chondroma has extended into the orbit from the septum, and myxoma has extended into the orbit from the frontal sinus. A malignant growth extending from the nasal accessory sinuses is, fortunately, not as common as one might expect, considering the high percentage of persons who suffer from chronic and recurrent sinusitis.

A metastatic malignant growth secondary to tumor of the stomach, the adrenal glands, the kidney, or the long bones of the body has been reported frequently as the cause of unilateral exophthalmos. This growth, naturally, is recognized as secondary, though the fact that it is so does not in any way change the seriousness of the condition. In Lowenburg's [63] case of neuroblastoma of the adrenal gland and in Van Duyse
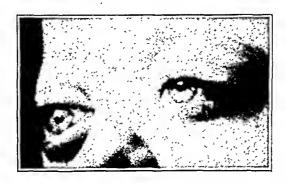
Fig. 28.—Retrobulbar sarcoma in a child (O'Brien).

and Marbaix's [64] case of hypernephroma, the condition was typical of the rare forms. Retrobulbar carcinoma has been seen secondary to adenocarcinoma of the stomach, and retrobulbar sarcoma has been seen resulting from osteogenic sarcoma of the tibia, and these cases are not exceptions.

DIFFERENTIAL DIAGNOSIS

Trauma, inflammatory conditions and neoplasm are the three conditions which present the greatest problems in arriving at a correct diagnosis in any case of unilateral exophthalmos under consideration. The history is of tremendous importance, especially in the consideration of some of the inflammatory possibilities. The rate of progress of the exophthalmos and the age of the patient at its onset will be of assistance in other cases. Pain is a characteristic symptom in some of the cases

63. Lowenburg, H.: Personal communication to the author, from the Mount Sinai Hospital, Philadelphia, 1936.

64. Van Duyse, D., and Marbaix: Métastase eithmoïdo-orbitaire d'un hypernéphrome latent. Arch. d'opht. **39**:396 (July) 1922.

SOCIETY NEWS

International Committee for the Protection of Eyesight and for the Prevention of Blindness.—During the five years of its existence the International Committee for the Protection of Eyesight and for the Prevention of Blindness has established branch committees in almost all parts of the world. The organization has dealt with the prevention of industrial accidents, with trachoma and with the erection of schools for children with reduced or impaired vision. The latest report of the committee reveals that there are about 6,000,000 blind persons in the world. The chief causes of blindness are injuries, glaucoma, heredity and infection. The committee makes provisions for the education of the unfortunates so that they may work and supports them in their activities.

National Society for the Prevention of Blindness.—The National Society for the Prevention of Blindness has issued its annual warning to parents regarding the risk to children's eyesight through the careless use of fireworks on the Fourth of July. A study of accidents due to fireworks, made two years ago, disclosed a total of 6,940 injuries which were serious. In the American schools for the blind there are now nearly 500 children who lost their sight as a result of accidents, chiefly through the use of fireworks and air rifles.

Correspondence

"TREATMENT OF RETINAL GLIOMAS BY THE FRACTIONATED OR DIVIDED DOSE PRINCIPLE OF ROENTGEN RADIATION"

To the Editor:—In a paper entitled "Treatment of Retinal Gliomas by the Fractionated or Divided Dose Principle of Roentgen Radiation," publihsed in the November 1936 issue (ARCH. OPHTH. **16**: 734), my collaborator and I stated:

Verhoeff's patient . . . received a "suberythema dose" of lightly filtered roentgen radiation to the eye once a week for three weeks.

This statement is not in accord with Verhoeff's report, which reads:

Dr. Liebman gave the first treatment on the same day, a sub-erythema dose through a 5 mm. aluminum filter. After this, similar treatment was given once a week for three weeks, and on June 6th, June 27th, August 1st, November 1st, 1918, and on January 17th, 1919.

We admit the error in quotation and gladly take this opportunity of correcting it.

A. B. REESE, M.D., New York.

Abstracts from Current Literature

Anatomy and Embryology

CONNECTIONS OF THE RODS AND CONES AT THE LEVEL OF THE EXTERNAL PLEXIFORM LAYER. F. FERNÁNDEZ BALBUENA, Arch. de oftal. hispano-am. 36: 337 (July) 1936.

After reference to the exceptional interest attached to the external plexiform layer as the site of the connections between the first and second retinal neurons, to the investigations hitherto made with modern methods of staining and to certain prevailing errors due to artefacts, Balbuena presents a remarkable study illustrated with a number of beautiful photomicrographs of sections of the external plexiform layer in the owl, rabbit, monkey and man; most of which were stained according to the method of Golgi. The article is posthumous; Balbuena died on March 4, 1936.

He maintains that the terminal "rod" spherules are mostly in contact with the bases of the cones, which they surround; he describes the topography, with its variations, of the "cone" bases, which are situated external to the external plexiform layer, with the connections of their divergent and convergent fibrils. He also describes "sinaxic bipolar cells," a new variety of horizontal cells, which receive impulses from the rods and cones and are connected with the external endings of Cajal's "rod" bipolars, the ascending fibers of the so-called horizontal cells. These cells he considers as forming an intermediate neuron (the proper second neuron), collecting luminous impulses from the cones and rods, whereas Cajal's "cone" bipolars receive excitations from only the foveal cones. He finally describes the following sublayers of the external plexiform layer: (1) a dense plexus of fibrils uniting the bases of the cones; (2) a superficial dendritic plexus, described by Cajal, formed by fibrils from the internal horizontal elements; (3) Cajal's internal dendritic plexus with an interlacing of large branches from the large horizontal cells; (4) horizontal ramifications of the cone bipolars, a dense plexus of centrifugal fibers, described by Cajal, and the terminal branches of the axis-cylinders of the horizontal cells. In the midst of the last two layers he observed the ascending branches of Cajal's rod bipolars and conical dilatations of the sinaxic bipolars, the bases of which appear above the external plexiform layer.

C. E. FINLAY.

Bacteriology and Serology

ANTIGENIC PROPERTIES OF THE CORNEAL PROTEIN. S. RINALDI, Ann. di ottal. e clin. ocul. 64: 691 (Oct.) 1936.

Rinaldi describes the use of intradermal tests made with a corneal emulsion on 200 patients with various types of keratitis and on 150 normal persons. The material was prepared by using one cornea to 10 cc. of physiologic solution of sodium chloride, 0.1 cc. of which was used for the intradermal test. Only 3 patients with keratitis, including

2 with serpiginous ulcer and 1 with phlyctenulosis, and 2 normal subjects, gave positive intradermal reactions. This is added evidence that the corneal protein possesses few or no antigenic properties and seems to dispose effectively of various theories, such as that of Elschnig, which attempt to explain interstitial keratitis and other conditions on the basis of autosensitization to the corneal proteins. A bibliography is included.

S. R. Gifford.

Biochemistry

Recent Observations on the Biochemistry of the Lens. Dorothy R. Campbell, Brit. M. J. 2: 1133 (Dec. 5) 1936.

This is a review of work which has been done on the biochemistry of the lens during the past three years. New methods for producing experimental cataract and their results are described under the following headings: (1) a disturbance of carbohydrate metabolism, (2) a deficiency of vitamins, (3) investigation of hereditary cataract in animals and (4) the administration of toxic substances.

In addition, general metabolic changes which may be associated with cataract have been investigated, and new substances in the lens have been identified.

This survey has been most carefully done and gives a valuable outline of present knowledge. The contents cannot easily be given in a review. Those interested will be well repaid by a study of the original article.

The author concludes as follows: "An excess of sugar or deficiency of vitamin, a disturbance of calcium metabolism, or loss of cystine from the lens, are all very likely factors in the causation of cataract, and even the effects of rare toxic substances have acquired an unexpected significance. Not only new facts, but those with which we are already familiar, are assuming a more orderly arrangement."

A. Knapp.

The Magnesium Content of Ocular Tissues. R. Wolff and A. Bourquard, Compt. rend. Soc. de biol. 124: 319, 1937.

The glycolytic action of the retina is the highest in the body, and magnesium is the coferment required for glycolysis. The distribution of magnesium in the tissues may possibly be correlated with functional activity. The magnesium content of the retina is high (90 mg. per hundred grams of dry tissue) but inferior to that of muscle. The pigment layer contains somewhat less magnesium than the retina, and the optic nerve, only half as much.

J. E. Lebensohn.

Comparative Ophthalmology

Retinal Localization in Relation to Dorsoventral Polarity of Black Coloring in Axolotl. V. Vilter, Compt. rend. Soc. de biol. 124: 47, 1936.

The axolotl (larval salamander) exhibits a chromatic adaptation to its background in which the dorsal coloration regularly fades out ven-

trally. A blinded animal becomes progressively darker but maintains the normal polarity. However, after 180 degree torsion of the seeing eyeballs was performed surgically, the animal's color became uniform the next day, and later the usual polarity was reversed. With only one eye turned, a contralateral change in pigmentation ensued. Hence the visual field appears related to the neuro-endocrine regulation of the melanocytes.   J. E. LEBENSOHN.

THE FOVEAL VISION OF FISH. M. F. CANELLA, Compt. rend. Soc. de biol. **124**: 405, 1937.

Observations on fresh water fish blinded in one eye confirm the conclusion indicated by the author's similar investigation on mammals: Blinding of one eye simply reduces the total visual field without appreciably affecting third-dimensional perception.   J. E. LEBENSOHN.

Cornea and Sclera

A METHOD FOR MAKING CASTS OF THE HUMAN CORNEA. C. L. STEVENS, Am. J. Ophth. **19**: 593 (July) 1936.

Stevens describes a method of making casts of the cornea with negocoll. This is melted or dissolved in water and allowed to cool at 40 C. The eye is anesthetized; the patient is instructed to look at a mark on the ceiling, and the negocoll is applied to the cornea by means of an aluminum container applied to the globe. The inner canthus should be marked on the container tube. The negocoll sets in about five minutes and is then removed and immersed in cool water and properly labeled. From this a cast is made of French's "Regular" dental plaster, the identifying mark at the inner canthus being preserved. ·   W. S. REESE.

INFLUENCE OF VITAMIN A ON THE REGENERATION OF CORNEAL EPITHELIUM. E. HEINSIUS, Arch. f. Ophth. **136**: 103 (Sept.) 1936.

Clinical observation has shown that vitamin A applied locally has a favorable influence on the healing of wounds. Heinsius produced complete corneal abrasions on both eyes of the rabbit and watched the regeneration of the epithelium under the influence of cod liver oil or pure vitamin A, which was instilled regularly into the conjunctival sac of one eye while the other eye received only paraffin oil. The defects healed more quickly in the eyes which were treated with preparations of vitamin A. The vitamin D contained in cod liver oil had no effect on the regeneration of the epithelium.   P. C. KRONFELD.

HISTOLOGIC-ANATOMIC RESEARCH ON TRUE MEGALOCORNEA GLOBOSA. B. KAYSER, Klin. Monatsbl. f. Augenh. **96**: 721 (June) 1936.

Kayser concludes with this paper his series of publications of his research on megalocornea. The following definite information was gained from the histologic-anatomic examination of one eyeball:

1. Glaucoma played no rôle in the development of the megalocornea.
2. It is certain that one form of true, uncomplicated megalocornea exists, in which the changes are limited to the anterior segment of the

eyeball, especially to the shape of the cornea, with later changes of the chamber angle and ciliary body. No inflammatory or degenerative processes exist by which secondary enlargement of the cornea could be explained as a result of softening or extension.

3. True megalocornea must be strictly distinguished from "secondary megalocornea," which is the result of pathologic processes. True megalocornea is identical with Horner's megalocornea globosa.

4. No findings have been made which would cast a doubt on the view that megalocornea is a developmental anomaly.

5. Macrophthalmos and megalocornea are two different conditions; enlargement of the cornea is a symptom common to both.

<div align="right">K. L. Stoll.</div>

Experimental Pathology

Production of the Shwartzman Phenomenon in the Cornea. G. Fabiani and A. Gauthier, Compt. rend. Soc. de biol. 124: 51, 1937.

The hemorrhagic allergic phenomenon of Shwartzman occurs readily in the conjunctiva and iris, though not in the cornea. This immunity, however, is solely based on the normal corneal avascularity. An infected corneal ulcer in a rabbit terminated in a highly vascularized leukoma. When a bacterial filtrate was injected, first in the corneal tissue and the following day intravenously, a hemorrhagic extravasation in the cornea appeared.

<div align="right">J. E. Lebensohn.</div>

The Nature of Disk-Shaped and Ring-Shaped Corneal Opacities. Y. Sugita, Arch. f. Ophth. 136: 52 (Sept.) 1936.

In Sugita's fundamental experiment a deep but nonperforating corneal wound about 1.5 mm. in diameter was produced with the galvanic cautery and concentrated sulfuric acid applied to its floor. The result was the development of a dense disk-shaped corneal opacity which, according to the author, was analogous to the periodic precipitation in the form of concentric rings which takes place in gelatin by the gradual diffusion toward each other of two types of ions previously added to different portions of the gelatin (Liesegang's rings). The clinical and the pathologic picture of this experimental corneal opacity are the same as those of disciform keratitis in man. In the latter case micro-organisms apparently are capable of eliciting biochemical processes similar to those in Liesegang's experiment. The same consideration may apply to other types of keratitis in which disk or ring-shaped opacities occur.

<div align="right">P. C. Kronfeld.</div>

Effect of Nephrotoxin on the Eye. S. Yonechi, Arch. f. Ophth. 136: 312 (Dec.) 1936.

Zur Nedden was the first to report retinal changes after injection of a specific nephrotoxin into the carotid artery. Since then, in 1908, Japanese investigators under the direction of Mita (the University of Tokio) found that retinal and renal proteins are closely related immunologically. Yonechi, a pupil of Koyanagi, was chiefly interested in the

effect of nephrotoxins on the pigment epithelium. By repeated intraperitoneal or subcutaneous injection of an emulsion of rabbit kidneys into goats serums were obtained which, if injected into rabbits, produced, clinically and pathologically, diffuse glomerulonephritis (Masugi: *Beitr. z. path. Anat. u. z. allg. Path.* 92: 429, 1934). The injections were made into the common carotid artery. Ligation of the artery after the injection was never necessary. The effect on the eye of the side given the injection appeared in the form of chemosis, exophthalmos and gray streaks and patches in the retina. Pathologically, small accumulations of coagulated protein were found in the retina, with abnormal secretory activity of the pigment epithelium and secondary degenerative changes in the latter and in the adjoining retinal layers. "The nephrotoxin acts chiefly on the pigment epithelium."

P. C. KRONFELD.

General Diseases

DIABETES AND THE EYE. A. E. GOLDFEDER and M. A. KOPELOVITS, Sovet. vestnik oftal. 9: 798, 1937.

In the Central Ukranian Institute of Endocrinology a study was made of 500 diabetic patients.

Lowered tonus of the eyeball was found in 53 per cent of the patients. In 70.6 per cent of the young patients disturbance of accommodation was observed. Refractive errors were of myopic and of hypermetropic character. Cataracts were found in 7.3 per cent. In the untreated diabetic persons the cataracts ripened very fast, while in the treated ones the ripening of the cataracts usually took many months. There were always vacuoles beneath the anterior capsule, also *Wasserspalten* (water slits). Extraction of cataract presented no difficulties provided that the patient's blood and urine were rendered free from sugar and acetone.

Diabetic retinitis (hemorrhagic type) was observed only in 1.8 per cent, mostly in elderly patients. Insulin therapy caused increase of hemorrhages in a few patients, so the authors believe that it should be given cautiously.

Since xanthelasma was found in 1.4 per cent, the authors think that it may be connected with disturbance of carbohydrate metabolism. In 35 per cent there was weakness of ocular muscles or absence of convergence; only one patient suffered from paresis of the sixth nerve. In 14 per cent there was anisocoria, and in 6.6 per cent Stellwag's and Graefe's signs were present.

O. SITCHEVSKA.

ROENTGENOTHERAPY IN LUPUS OF THE CONJUNCTIVA AND CORNEA. F. B. GOLDBERG, Sovet. vestnik oftal. 9:893, 1937.

A girl aged 19 suffered from lupus of the face six years ago. The condition improved under treatment with roentgen rays and tuberculin. Four years ago the left eye became inflamed, and the inflammation persisted despite routine therapy. Examination revealed thickening of the conjunctiva of the lids and of the fornices with solitary small grayish nodules. The bulbar conjunctiva presented more advanced changes. It was thickened and reddish gray, with an abundance of grayish nodules; it was covered with a granulation tissue that extended to the

periphery of the cornea. The granulation tissue was heaviest in the upper sector, resembling pannus crassus, and showed a number of gray nodules. Histopathologic examination showed that the nodules were tuberculous granulomas consisting of epithelioid and giant cells with some fibrous tissue.

Since surgical intervention was impossible because of the extensiveness of the process, roentgenotherapy was decided on; ⅛ of 1 H.E.D. (unit skin dose) was administered every seven to ten days. The treatments (seven) caused marked aggravation of the symptoms, and vision was reduced from 0.2 to 0.06. This persisted for three months, with a further reduction of vision to perception only of light. A larger dose of roentgen rays (¼ of 1 H.E.D.) was given, and after four treatments the symptoms began to subside. In a few months fine scars formed in the conjunctiva, the pannus absorbed, and vision improved, but only to 0.05 because of diffuse central corneal opacities. Thus a larger dose of roentgen rays proved to be the means of curing this rare lupus, while the initial smaller dose caused aggravation of the process with irreparable loss of vision.

O. Sitchevska.

Glaucoma

Clinical Measurement of the Ocular Tension. P. Bailliart, Ann. d'ocul. 173: 945 (Dec.) 1936.

Following a recent communication read at a meeting of the Ophthalmologic Society of Paris, in which Mérigot of Treigny justly defended the operative treatment of glaucoma, the question has naturally been raised again as to the most opportune time for operation. Is this when hypertension commences? What is the highest limit of physiologic tension that is known?

Morax, an authority whom one always likes to quote, wrote in 1921: "One considers as normal, that is, having no harmful consequences, a tension between 15 and 28 mm. of mercury. In fact, since I have examined the globe with the Schiötz tonometer I have never met with glaucomatous symptoms in eyes having a tension less than 30 and have never considered as pathologic any aggravation in the tension of glaucomatous eyes up to 28. Does this signify that the physiologic ocular tension lies between 18 and 28 mm. of mercury?"

Bailliart gives in detail his experiments with the tonometer on patients in his clinic. He divides the cases into three groups. In the first group the ocular tension remained as it was when first taken, the needle moving between 25 and 23. In the second group the needle continued to move but tended to move downward, oscillating between 24 and 22, 23 and 21, etc. In the third group the reverse occurred; the needle continued to move but moved upward, oscillating between 34 and 30, 36 and 32, etc.

Too much importance must not be placed on the tonometer. Although it is an excellent instrument, its value is relative. To establish the diagnosis, prognosis and treatment, one must not depend on figures but search for symptoms, and to relieve symptoms the cause must be found.

S. H. McKee.

INTRA-OCULAR TENSION AFTER PARACENTESIS IN EYES WITH NORMAL TENSION, CHRONIC SIMPLE GLAUCOMA AND UVEITIS WITH HYPOTONY. O. BARATTA, Arch. di ottal. 43: 211, 1936.

Ten eyes with cataract but normal tension, eight with chronic simple glaucoma and nine with anterior uveitis and hypotony were employed. In the eyes with normal tension a rise of tension amounting to from 3 to 5 mm. was found after three hours, which was replaced after one or two hours by slight hypotony, persisting as a rule for twenty hours. This was followed by a second slight rise of tension, persisting until from thirty-two to forty-five hours after the paracentesis. In the glaucomatous eyes a rise of tension of from 10 to 16 mm. above the original tension was observed after three hours. In five of the eight eyes this persisted until six hours after the paracentesis, after which a fairly rapid fall occurred to figures below the original tension, with a return to the original tension after forty-eight hours. In three eyes there was a gradual fall to the original tension, beginning after three hours and never going below that point. In six of the nine eyes with uveitis and hypotony, the tension reached only 1.5 mm. below the original tension after three hours, rising slowly to the original figure after from three to four days. In three of the nine eyes there was a slight increase of tension above the original figure, followed by prolonged hypotony like that seen in the other eyes with uveitis. In this group of eyes a disturbance of the mechanism for the formation of aqueous must be assumed, while in the normal and glaucomatous groups the behavior of tension was analogous to that of cerebrospinal fluid pressure after lumbar puncture or of arterial pressure after removal of blood.

S. R. GIFFORD.

Injuries

INJURIES OF THE EYES CAUSED BY SMALL SHOT. P. ILLÉS, Szemészet 70: 211, 1935.

Within the field of bullet wounds of the eye, those made by small shot, especially while the patient was hunting, occur most frequently. The entering course of small shot is always sagittal. In most cases only a single shot has entered, but the injury may have such serious consequences as retinal hemorrhage and detachment of the retina. Often the shot remains in the eye. Double perforation of the eyeball may occur, the shot remaining in the orbit, where it may cause injury or atrophy of the optic nerve.

The clinical notes for three of the author's cases are given in full. In the first case there was a superficial injury of the cornea, which healed readily, with consequent full acuity of vision. In the second case there was a hemorrhage into the vitreous, and an attack of glaucoma occurred. In this case healing took place within four days, and acuity of vision was normal. In the third case the shot lodged in the sclerotic wall, leaving no serious functional disturbance. Six months later the eye showed a permanent scotoma but no other damage.

The prognosis in cases of wounds made by small shot and double perforation may be favorable, but the injury may lead to serious con-

sequences, causing shrinking of the eyeball following iridocyclitis. It should be a rule that small shot should not be removed unless there is an inflammatory reaction.

N. BLATT.

Lens

EMBRYONAL, FETAL, ZONULAR AND CENTRAL CATARACT. G. FAVALORO, Ann. di ottal. e clin. ocul. 64: 721 (Nov.) 1936.

The older literature concerning congenital central (nuclear) cataract is reviewed, as well as that based on modern findings with the slit lamp. A case of bilateral embryonal nuclear cataract is described, the opacity being from 3 to 4 mm. in diameter and corresponding to the size of the lens in the fifth fetal month. In a second case each lens showed three zones of opacity: a central opacity, the most dense, corresponding to the embryonal nucleus; a more peripheral grayish zone with a sharply outlined border, corresponding to the fetal nucleus, and six spokes of opacity radiating from this zone to the periphery of the lens. Signs of defective development and of rickets were present. In a third case only the fetal nucleus was involved, but three layers could be distinguished. The most central was very dense, triangular and corresponded in size only to the region of the anterior Y suture. The second and third were less dense and of a powdery appearance, corresponding to the middle and late periods of fetal life. In a fourth case one eye presented a total cataract, but with a denser central opacity corresponding to the embryonal nucleus, while in the other eye only the fetal nucleus was involved. From these observations and others in the literature Favaloro would distinguish the zonular form of nuclear cataract in which all the zones of opacity are confined to the fetal nucleus from the more usual zonular, or perinuclear, cataract. The greater density of the most central portion in his nuclear cataract indicates an origin of the opacity very early in fetal life and in the most central part of the lens, in contradistinction from zonular perinuclear cataract, which begins in postnatal life or late in fetal life. It is also possible to distinguish, according to the size of the opacity, between embryonal cataract, which develops during the first four months of intra-uterine life, and fetal cataract, which develops later in fetal life. If the process of opacification continues until the end of fetal life, a total cataract will result.

S. R. GIFFORD.

REPAIR OF CAPSULE WOUNDS BY THE SURVIVING LENS. A. BAKKER, Arch. f. Ophth. 136: 333 (Dec.) 1936.

Injury of the anterior lens capsule in man causes, as a rule, a progressive cataract or complete absorption of the lens, while in rabbits and in a few other animals the wound is closed by formation of fibroblast-like cells, which are overlaid by regenerated lens capsule. The process remains confined to the site of injury, and the transparency of the bulk of the lens is not disturbed. The question of the origin of the fibroblast-like cells has not been solved. Schirmer believed they were derivatives of the lens epithelium. Bakker studied these processes on the surviving lens (*Arch. f. Ophth.* 135: 581, 1936; 136: 166, 1936) and found that they were essentially the same as in vivo. Since the

only type of cells present in the surviving lens are cells of the capsular epithelium the ectodermal origin of the cells which repair capsular wounds in rabbits is now certain.

<div align="right">P. C. KRONFELD.</div>

Lids

ETIOLOGY AND PATHOGENESIS OF CHALAZION. G. SOLIGNAC, Ann. d'ocul. **174**: 108 (Feb.) 1937.

Even though chalazion is known as a benign disease and therefore of secondary importance from the clinical point of view, it has been the subject of numerous researches. Its histologic structure particularly has been studied. Even yet, however, there is considerable difference of opinion as to how certain characteristics of the chalazion should be interpreted. Bacteriologic investigation has not given satisfactory results, and the pathogenesis still remains obscure.

Solignac goes into considerable historical detail from 1858, when Sichel attributed the condition to a microbic origin, to 1932, when Bailey gave a general review of the anatomy, pathology and surgery. The histologic examination is given in detail, with two illustrations, also the results of experimental inoculations and bacteriologic examinations.

He concludes from this histologic study that he is in accord with Shall in the opinion that the chalazion is the result of a chronic inflammatory process with phagocytosis in which the bacterial infection does not play a primary rôle but is secondary, and that the condition is relatively rare. The term *granulome stéatophagique* employed by Levaditi seems justified. A bibliography is given.

<div align="right">S. H. McKEE.</div>

Methods of Examination

THE IMPROVED FIVE POINT VISUAL ADAPTOMETER FOR MEASURING SENSITIVENESS TO LIGHT. A. BIRCH-HIRSCHFELD, Klin. Monatsbl. f. Augenh. **97**: 433 (Oct.) 1936.

The efficiency of the five point adaptometer devised by Birch-Hirschfeld for measuring the sensitiveness to light was shown during the war. It was used also for scientific research, for example, by Ira O. Park in his studies on vitamin deficiency. This apparatus has been improved by the author in many respects. The chief principle remains unchanged: Five points of light are arranged in such a way that two points placed vertically on the left side are brighter and two on the right side are darker. This arrangement facilitates accurate observation of the fifth point, located in the center. In the improved apparatus the gray graduated wedge is replaced by a series of graduated gray glasses. They are fitted on a disk in a circle, so that by inserting them behind the five points the brighter points can be made to appear darker than the central point and the darker points brighter than the central point. A detailed description of the apparatus and the technic is given and illustrated by pictures and a table. Satisfactory results were obtained by Birch-Hirschfeld with this improved apparatus in examination of a large number of airmen at the University Eye Clinic of Königsberg. Germany.

<div align="right">K. L. STOLL.</div>

Neurology

OCULAR SYMPTOMS OF SUPRASELLAR MENINGIOMAS. E. HARTMANN and L. GUILLAUMAT, Ann. d'ocul. **174**: 1 (Jan.) 1937.

In its development, suprasellar meningioma presses on the optic nerve and the chiasm and by so doing causes visual disturbance, which is one of the first clinical signs and one of the important elements in the prognosis. The condition is often confused with retrobulbar neuritis of toxic or infectious origin. Surgical intervention offers the only possible treatment and gives good results. Vision is improved, and, as meningioma is histologically benign, it does not become generalized, while a local recurrence, if it does take place, comes on slowly.

Cushing stated: "When the question 'what's wrong with you?' is put to adult patients who are referred to a neurosurgical clinic, the answer 'my sight is failing' is probably, in these latter days, far more often given than any other." Failure of central vision and contraction of the peripheral visual field should direct one to immediate examination of the fundus.

In this article a summary of the findings for twenty-one patients is given. The diagnosis was verified microscopically and histologically in all the cases. The article is illustrated with drawings and tables, and a bibliography is given.   S. H. McKEE.

RELATIONS BETWEEN HEMIANOPIA AND OPTOKINETIC NYSTAGMUS. J. OHM, Arch. f. Ophth. **136**: 341 (Dec.) 1936.

In a large number of cases of hemianopia and with his very accurate method of nystagmography Ohm has studied optokinetic nystagmus. His study confirms his previously expressed opinion that a lesion con-confined to the visual pathway does not affect optokinetic nystagmus, and also, at least in principle, Cord's view that optokinetic nystagmus toward the blind side becomes disturbed if the efferent as well as the afferent visual radiation is affected. Ohm, however, has seen, and quotes others who have seen, exceptions to the latter rule.

                    P. C. KRONFELD.

TUMORS IN THE REGION OF THE HYPOPHYSIS. M. BARTELS, Klin. Monatsbl. f. Augenh. **97**: 185 (Aug.) 1936.

Bartels selected for his report seventeen cases of tumor in the region of the hypophysis, which he observed to show the importance of ophthalmoscopic and allied examinations of patients suffering from apparent anemia, "neurasthenia" and other seemingly constitutional diseases. Critical considerations are given for each case, and a résumé of the ocular and general disturbances found is offered. Each of these disturbances, including a number of instructive deviations from the usual pathologic aspects, is discussed critically in detail.

In concluding, Bartels states that his observations furnished only this definite general conclusion: A reliable prognosis could be made in no case, nor was it possible to foretell definitely whether an operation was indicated or not. Every case of this type should be referred for observation to some "central" or clearing house, a custom which

is being practiced by some groups of surgeons in Germany. Thus the best results would be obtained, and the question of surgical procedures might be considered carefully. Bartels points out a number of errors leading to wrong diagnoses, e. g., in cases in which a tumor is absent which was thought to be present. In this connection he mentions physiologically abnormal growth in males during the period of development in puberty which may resemble adiposogenital dystrophy. One of his cases belonged to this group; the patient presented a transient hormonal disturbance during puberty. Conditions of this kind may be based on familial heredity and appear during puberty in persons who remain perfectly normal afterward during their whole life. The absence of ocular symptoms in a suspected case at any time would not entirely exclude the presence of a tumor of the hypophysis, yet a wrong diagnosis would cause unnecessary excitement in the family.

K. L. STOLL.

Orbit, Eyeball and Accessory Sinuses

THE OCULOCARDIAC REFLEX AND SUGAR METABOLISM. G. PORTO-GHESE, Arch. di ottal. 43: 181, 1936.

Literature on this reflex and also on the oculovasomotor reflex is reviewed. Both are produced by pressure on the globe and seem to depend on central stimulation of the vegetative nervous system as a result of afferent impulses received through the fifth nerve.

Portoghese estimated the sugar in the capillary and venous blood of eighteen normal persons before and for an hour after digital pressure on both globes, maintained for one minute. In all these persons he observed a positive oculocardiac reflex of the type considered normal. The slowing of the pulse was not over twelve beats per minute. In all cases an increase of blood sugar was noted which amounted to from 0.06 to 0.3 per cent. This occurred within from ten to twenty minutes. In a few cases a second lowering of the pulse occurred· after from twenty to thirty minutes, which was accompanied by a second rise in the blood sugar. The difference between the capillary and the venous glycemia was very slight (from 0.02 to 0.09 per cent), indicating that the rise in the blood sugar was not due to an increased amount of insulin in the blood. The conclusion seemed justified that the rise in the blood sugar was due to stimulation of the sympathetic-adrenal mechanism. There was no constant relation between the degree of bradycardia and the amount of hyperglycemia. A bibliography is included.

S. R. GIFFORD.

The Pupil

COMPARISON OF SUPRARENIN BITARTRATE AND COCAINE PLUS EUPHTHALMINE AS MYDRIATICS FOR ELDERLY PATIENTS. L. T. POST, Am. J. Ophth. 20: 33 (Jan.) 1937.

Post reviews the work of Horner and Bettman on mydriasis from suprarenin bitartrate. He then compares the action of suprarenin bitartrate, 2 per cent, with that of cocaine, 1 per cent, plus euphthalmine,

1 per cent, the former being instilled in the left and the latter in the right eye of each of ten patients. He found that: (1) suprarenin acted faster and more powerfully, though in a small percentage of cases it did not dilate the pupil; (2) cocaine plus euphthalmine caused an increase in tension in four cases; (3) the mydriasis was much less easily controlled with physostigmine in the cases in which suprarenin was used, and in six of these pain supervened whereas there was no occurrence of pain in the cocaine-euphthalmine group. A number of incidental observations were made. Post concludes that cocaine plus euphthalmine is in general preferable as a mydriatic for ophthalmoscopic purposes in elderly patients.

W. S. REESE.

PUPILLARY ACTIVITY OF THE DIENCEPHALON AND MESENCEPHALON. E. CLAES, Compt. rend. Soc. de biol. 123: 1009, 1936.

Confirming Magoun and Ranson's observations (*Arch. Ophth.* 13: 791 [May] 1935), Claes finds that faradic excitation of the pretectal zone (the posteromedial portion of the dorsal thalamus) induces bilateral miosis, generally with an associated downward movement of the globes and closure of the lids. Stimulation of the corpora quadrigemini, especially the posterior tubercles, causes moderate mydriasis, unmodified by section of the cervical sympathetic nerve but abolished by a cut separating the anterior from the posterior tubercles. An independent but more marked mydriatic action follows stimulation applied to the anterior cut surface of the internal capsule inferomedially. These mydriatic actions work through inhibition of the Edinger-Westphal nuclei; extreme miosis results from section of the brain stem anterior to the third nerves. The miosis of sleep is based on this mechanism.

J. E. LEBENSOHN.

Physiologic Optics

A STUDY OF TWO HUNDRED AND EIGHTY-EIGHT PATIENTS EXAMINED WITH THE OPHTHALMO-EIKONOMETER. C. BERENS, Brit. J. Ophth. 21: 132 (March) 1937.

Berens describes the ophthalmo-eikonometer and the method of examining eyes with the instrument. The patients are classified according to age and sex; their symptoms are described, and the results obtained are tabulated. The relations of heterophoria and emmetropia to aniseikonia are considered. The following conclusions are given:

A study of 288 patients reveals no apparent significance in the age or sex of the patients for whom iseikonic lenses were prescribed. However, the percentage of females was slightly higher than that of males. A majority of the patients were between the ages of 25 and 40.

Apparently there are no pathognomonic symptoms which are complained of by patients with aniseikonia or which seem to be relieved by wearing iseikonic corrections. The most typical symptoms which seem to be relieved are asthenopia and headaches, especially when these symptoms are aggravated by reading.

In this series of cases there seems to be a tendency for the smaller image to be in the right eye and for the differences to be found more frequently in the horizontal than in the vertical meridian.

Apparently there was definite relief from symptoms in 68.7 per cent of the 160 patients who wore iseikonic lenses, and half of those who reported improvement had marked or nearly complete relief.

At present it is impossible to determine whether the factor which is measured with the ophthalmo-eikonometer and corrected by iseikonic lenses is aniseikonia alone.

Much scientific research with many hundreds of clinical experiments and careful analyses of statistics must be carried out before the exact place of aniseikonia in causing ocular symptoms can be conclusively evaluated.

W. ZENTMAYER.

Physiology

BLUE ARCS OF THE RETINA. H. PIÉRON, Compt. rend. Soc. de biol. 124: 523, 1937.

Audubert and Levy demonstrated that emission of ultraviolet rays followed stimulation of the sciatic nerve of the frog. Hertel found the cones of the frog's retina to be directly sensitive to light rays as short as 2,260 angstrom units. Piéron consequently suggests that the blue arcs may be due to this emission. Viewing ultraviolet rays produces no phenomena, since the ocular media absorb the effective rays.

J. E. LEBENSOHN.

OPTOKINETIC ELICITATION OF VERTICAL DEVIATIONS. H. BURIAN, Arch. f. Ophth. 136: 215 (Nov.) 1936.

Patterns consisting of black and white stripes were mounted on two drums revolving around horizontal axes. If these patterns were presented to the observer by means of the haploscope and both patterns moved in the same direction, the observer's eyes showed vertical opto-kinetic nystagmus. If one pattern was moving downward and the other upward, the eyes of the observer remained still. If during this procedure the observer also had to overcome a vertical prism equivalent to his vertical ductions, transient vertical diplopia resulted. These observations represent further evidence for the existence of an independent innervational complex for vertical divergence. The biologic significance of this complex lies, according to Tschermak-Seysenegg, in the functional compensation of heterophoria.

P. C. KRONFELD.

DETERMINATION OF THE PRESSURE IN THE VENAE VORTICOSAE OF NORMAL HUMAN EYES. E. SEIDEL, Arch. f. Ophth. 136: 303 (Dec.) 1936.

The work reported in this paper represents the application of the principle of ophthalmodynamometry to the venae vorticosae. Ophthalmoscopic examination of the intra-ocular portion of these veins in albinotic but otherwise normal human eyes reveals definite narrowing of the outlets of the venous sinuses when very slight pressure is exerted on the globe. Seidel's interpretation of this phenomenon is that the pressure in these veins is, just like the pressure in the retinal veins, only very slightly higher than the intra-ocular pressure. The pressure in the choroidal capillaries can be only slightly higher (according to the author

about 30 mm. of mercury) than that in the veins and production of intra-ocular fluid by physical forces alone is impossible.

P. C. KRONFELD.

Refraction and Accommodation

"TWINCENTRIC" LENSES. F. A. WILLIAMSON-NOBLE, Brit. J. Ophth. 21: 116 (March) 1937.

Williamson-Noble describes a bifocal lens with the center of the reading portion displaced inward to allow for convergence. It is not necessary for the centers to coincide; they need be only on the same horizontal level. The lens may be made as a solid bifocal, but so made it has the disadvantage of producing a conspicuous reading segment, the upper border of which is clearly visible and may annoy the wearer. The lens may also be made to a fused bifocal. The disadvantage of such a lens is that there is some chromatic aberration in the reading segment. This is practically abolished by making up the lens in a slight tint.

W. ZENTMAYER.

THE DEVELOPMENT OF MODERN METHODS OF ESTIMATING REFRACTION. W. B. E. McCREA, Brit. J. Ophth. 21: 118 (March) 1937.

This is a historical article tracing the development of lenses and refraction from antiquity up to 1870. The title of the article is misleading.

W. ZENTMAYER.

Retina and Optic Nerve

RETROBULBAR INJECTIONS OF ATROPINE IN ARTERIOSCLEROTIC ATROPHY. F. C. CORDES, Am. J. Ophth. 20: 53 (Jan.) 1937.

Cordes briefly reviews the literature and reports a case of arteriosclerotic atrophy in which there was improvement following retrobulbar injections of atropine.

W. S. REESE.

RETINAL ANGIOSPASM IN TOXEMIA OF PREGNANCY AND HYPERTENSION. E. SELINGER, Am. J. Ophth. 20: 56 (Jan.) 1937.

Selinger discusses spasm of the retinal arteries and emphasizes the importance of differentiating functional and organic disease ophthalmoscopically. He gives the following summary.

"Spasms of the retinal arteries may be intermittent or continuous. The presence of intermittent spasms facilitates the diagnosis and makes it more certain. Only a segment of an artery or several large branches may be affected. They are an almost constant accompaniment of toxemia of pregnancy and, together with other retinal changes are of great prognostic importance. The ophthalmologist has an opportunity to study, under great magnification, the type of vessel concerned in hypertension. A study of these vessels will help in differentiating the form of hypertension amenable to treatment from that in which treatment is hopeless.

"Eyeground changes in nephritis are largely the result of arteriolar disease rather than the nephritis per se."

W. S. REESE.

KATHOLYSIS IN THE TREATMENT OF RETINAL DETACHMENT: A PRE-
LIMINARY NOTE. H. B. STALLARD, Brit. J. Ophth. 21: 35 (Jan.)
1937.

Stallard describes fully the ocular ionizer he employs and describes
in detail his technic in operating for retinal detachment. He gives the
following conclusions and summary:

"Katholysis in the surgical treatment of retinal detachment is of
value for the purpose of localizing the site of a retinal hole in relation
to the external surface of the sclera at the time of operation. The
cauterization produced leads to fine choroido-retinal scars which in my
opinion, may be adequate for sealing small holes and for tears in the
lower half of the retina but have insufficient tenacity for moderate and
large holes in the upper half of the retina.

"Up to date none of the serious immediate and late post-operative
complications which are seen in some cases treated by surgical diathermy
have been noted after katholysis."

The article is illustrated.

W. ZENTMAYER.

THE SITUATION OF RETINAL TEARS. SCHIFF-WERTHEIMER, Ann.
d'ocul. 174: 39 (Jan.) 1937.

In the course of the necessary ophthalmoscopic examination that
precedes surgical treatment in detachment of the retina, Schiff-
Wertheimer has observed certain signs that have helped in the localiza-
tion of the tear. These varied according to the age of the patient, on
one hand, and according to the refraction of the eye, on the other.

The cases have been divided into three groups: (1) detachment of
the retina in adult myopes (high or low), (2) detachment in nonmyopes
past middle age and (3) detachment in children or young nonmyopic
subjects.

In the first group, myopes of middle age, the tear is found in the
superior part of the retina, about the vertical meridian—for example,
in the right eye between 11 and 2 o'clock. In the second group, myopes
of advanced age, the tear is usually found around or near the level of
the horizontal meridian, most frequently at the temporal side between
8 and 10 o'clock. In the third group, nonmyopic young subjects, the
visual disturbance is frequently in the superior nasal field, correspond-
ing in a great number of cases to retinal disinsertion in the inferior
temporal quadrant—for example, in the right eye between 7 and 8
o'clock.

S. H. MCKEE.

SPASMS OF THE CENTRAL ARTERY OF THE RETINA. J. SEDAN and
G. E. JAYLE, Ann. d'ocul. 174: 73 (Feb.) 1937.

This article is part III, continued from the August and November
numbers of the Annales, and begins with the subject of "Prolonged
visual disturbance of spasmodic origin." This is subdivided into the
following topics: blindness or amblyopia with the picture of embolus
of the central artery or of its branches; blindness or amblyopia with
the picture of simple atrophy; blindness or amblyopia from fever, and
blindness or amblyopia from diverse causes.

These conditions are described in detail, the second one being subdivided again into atrophy by essential spasm, tabetic atrophy, atrophy from quinine and diverse toxemias, and hemorrhagic atrophy. Illustrative cases are described.

This study shows that the conditions described under the term "retinal spasm" may be placed in two distinct categories. In the first are found all the amblyopias or transitory blindnesses called spasmodic associated with embolus of the central artery. The second includes all the amblyopias and permanent blindnesses attributed to spasm with embolic conditions excluded. No comparison between the two groups can be made which would warrant any form of transition between the two.

The bibliography of the complete article is added.

S. H. McKee.

ANGIOSPASM OF THE RETINAL ARTERIES. L. MARUCCI, Ann. di ottal. e clin. ocul. 64: 671 (Oct.) 1936.

Marucci reviews the literature on spasm of the retinal arteries, his interest being in spasm of the smaller vessels rather than in complete closure of the central artery. He reports a remarkable case which resembles some cases reported by Castelli previously abstracted in the ARCHIVES. The patient was a woman of 38 in whom visual disturbances occurred at the seventh month of lactation. During this time the patient had nursed not only her own child but another infant. She had increased rapidly in weight for several years before and had noted menstrual irregularities. The visual disturbances were ushered in with nausea, dizziness and intense pain in the left eye. After three days vision was reduced to perception of movements of the hand. On the sixth day similar disturbances began in the right eye, and after five days vision was reduced to perception of light in this eye. The blood pressure showed only a slight elevation, and microscopic examination of capillaries showed slowing of the circulation in the nail fold, with tortuosity of the vessels. Otherwise the physical findings were practically normal. Each eye showed marked edema of the disk and surrounding retina, with large tortuous veins and small arteries. There was slight improvement in vision, with ability to count fingers, following the use of acetylcholine. Nineteen days after the onset the use of an estrogenic preparation was begun, 2,000 units being injected on alternate days. Vision began to improve rapidly and after twenty days of treatment had reaced 6/10 in each eye. The treatment was stopped, and vision at once began to diminish, decreasing to ability to count fingers in each eye. Treatment with estrogen was resumed, and vision improved to 10/15 in the right eye and 10/10 in the left after thirteen days. Further improvement occurred, so that five weeks later vision was normal in each eye. The right disk was pale, with blurry borders, while the left fundus was reported as normal.

Marucci believes the condition was due to reduction in the caliber of the retinal arteries due to spasm. While the vessels were not completely occluded, their volume was reduced. The attacks of headache and vertigo seemed to indicate spasm of other vessels. The association with lactation and menstrual disturbances and the marked improvement on treatment indicate ovarian dysfunction, though other glands were probably involved.

S. R. Gifford.

DIABETIC RETINITIS: A STUDY OF ONE HUNDRED AND FIFTEEN CASES. R. BRAUN, Arch. f. Ophth. **136**: 256 (Nov.) 1936.

The paper is based on thorough clinical studies of 770 diabetic persons, 115 of whom showed retinal changes. The diabetes in the latter patients was moderately severe and of long duration. The characteristic ophthalmoscopic features of the retinitis were *absence* of edema of the retina and of disk and vascular changes. Braun believes that the retinitis in such cases is caused by the diabetic disturbance of tissue metabolism, i. e., chiefly by the acidosis and ketonemia. Vascular changes and hypertension are, contrary to the common belief, not primary. In some of the cases the retinitis seemed to be related to renal insufficiency. There is no efficacious treatment for diabetic retinitis. The author has seen no sign that insulin exerts harmful effects on the normal or on the diseased retina.

P. C. KRONFELD.

THE HEREDITY OF HYALINE BODIES OF THE DISK. M. LEIMGRUBER, Arch. f. Ophth. **136**: 364 (Dec.) 1936.

To the five known families in which several members of one or two successive generations showed hyaline bodies (drusen) of the disks Leimgruber adds five more. None of the patients revealed signs of active or healed neuritis. Some brothers or sisters of patients with definite hyaline bodies showed slightly elevated, indistinctly outlined "pseudoneuritic" disks. For these, the presence of deeply situated and therefore invisible hyaline bodies is assumed. "Hyaline bodies may be the result of neuritis or the result of an inherited tendency toward the deposition of hyalin."

P. C. KRONFELD.

OCCURRENCE AND ETIOLOGY OF DETACHMENT OF THE RETINA: REPORT OF CASES. W. MEISNER, Klin. Monatsbl. f. Augenh. **97**: 289 (Sept.) 1936.

Meisner studied 180 eyes with detachment of the retina which came to his observation at the ophthalmic clinic of the University of Cologne between 1932, the year in which operations were introduced systematically for this disease, and 1935. He attempted to divide these cases into groups, eliminating those cases from his statistics in which intra-ocular or orbital tumors were the etiologic factors, those in which the eye presented transient detachment resulting from nephritic edema or exudation and those in which the detachment was caused by isolated inflammatory choroidal foci. The occurrence of tears is given due consideration, and mention is made that in 111 eyes, or 61.7 per cent of the 180, the detachment was cured. Furthermore, the following questions are discussed: whether the duration of a detachment may be estimated and to what extent recovery and function of the fovea depend on the time which elapsed between the observation of the first symptoms and the operation. Detachment due to contusion and other injuries was found in 26 eyes, 5 of which were emmetropic, the detachment occurring at the site of the injury. Detachment followed contusion in 19 eyes,

13 of which had been otherwise damaged or were myopic. Meisner thinks that detachment in myopic persons may be caused by contusions.

The location of the tears is discussed. Meisner found the tear after injuries in the temporal half of the eyeball, contrary to Gonin's experience. Indirect lesions and concussions led to detachment in 5 eyes, 4 of which were myopic; the second eye became afflicted spontaneously in 2 patients later on. In conclusion Meisner expresses his opinion as follows: The extent of the detachment allows no estimation regarding its duration; the size of the tear and the condition of the vitreous must be taken into consideration. An interval in which the eye is free from symptoms does not preclude a connection between injury and detachment.

Myopic and aged persons are especially subject to detachment, myopic persons being affected chiefly between the ages of 30 and 50, persons of advanced age, after 50 years of age. The higher the myopia, the greater the danger. Two groups of detachment may be distinguished in emmetropic persons less than 50 years old: those with spontaneous large tears in the temporal lower portion of the ora serrata and those with so-called inflammatory detachment.   K. L. Stoll.

TREATMENT OF ATROPHY OF THE OPTIC NERVE: REPORT OF CASES. H. Arruga, Klin. Monatsbl. f. Augenh. 97: 308 (Sept.) 1936.

Arruga treated a number of patients with tabetic atrophy of the optic nerve according to Lauber's therapy, which aims to increase the lowered blood pressure of these patients. Six case histories are reported, and the treatment is described. It consists of nourishing food, hypodermic injections of strychnine, caffeine, epinephrine hydrochloride, ephedrine sulfate and atropine sulfate, also sun baths and mountain climate, if possible. Vision was increased in three of the six patients and decreased in two; it remained stationary in a number of cases. Arruga made the following observations: The blood pressure of patients with tabetic atrophy of the optic nerve is generally low. Failure of vision of some of these patients may be caused by compression of the retinal capillaries in consequence of insufficient blood pressure in the intra-ocular arterial system. The course of the disease is favorably influenced by raising the general blood pressure and lowering the intra-ocular pressure. Similarly, patients with glaucoma show improvement as soon as the blood pressure rises, while they experience deterioration as soon as it decreases.   K. L. Stoll.

TREATMENT OF ATROPHY OF THE OPTIC NERVE WITH RETROBULBAR INJECTIONS OF ATROPINE SULFATE. V. I. Kosmin, Sovet. vestnik oftal. 9: 686, 1937.

During 1932-1934, thirty-five patients aged from 10 to 60 years, suffering from optic atrophy of various causes, were treated with retrobulbar injections of a 1: 1,000 solution of atropine sulfate. The injections were given every other day, the doses being 0.03, 0.05, 0.08 and 1 cc., about ten injections being made into each orbit. No complications were observed except dryness in the mouth. The intra-ocular tension

was unchanged. The visual fields were examined before and after the injections. The time of observation was from five to twelve months; a table gives the data.

Kosmin arrived at the following conclusions:

1. Retrobulbar injections of a 0.1 per cent solution of atropine sulfate brought about improvement of vision in 73.5 per cent of the cases.

2. Injections of atropine sulfate into the retrobulbar space of one eye resulted in improvement of vision in the other, untreated eye also, in 58.3 per cent of the cases.

3. In 41 per cent of the cases, enlargement of the visual field for white was noticed.

4. The injections of atropine sulfate did not cause dilatation of the pupil.

5. The increase of visual acuity was observed usually half an hour after injection, at which time a maximal increase of the temperature in the conjunctival sac was also registered. Thus, possibly active hyperemia plays a part in the result of the injections.

6. The results were more favorable in cases of postneuritic atrophy and in those of spasm of the retinal vessels than in cases of simple atrophy.

O. SITCHEVSKA.

SEMEIOLOGIC VALUE OF CHOKED DISK WITH TUMOR OF THE BRAIN AND NEURITIS OPTICA. D. GRIGORESCU, V. BUCUR and G. CONSTANTINESCU, Spitalul 55: 149 (April) 1935.

Mistakes are still occurring in the differential diagnosis of choked disk and papillitis. It is altogether wrong to talk about choked disk when but slight elevation of the optic papilla is present (up to 2 diopters). It is realized also that, with inflammatory optic neuritis, rather intense edema of the papilla may be present. To distinguish clearly between choked disk and inflammatory papillitis is a matter of decisive prognostic importance as well as a guide for treatment.

The authors had the opportunity of observing diagnostic errors in two patients. The first was a man 27 years of age; an oculist diagnosed choked disk. Neurologic examination disclosed no evidence of any disturbance of the central nervous system. The acuteness of vision had markedly decreased. The serum Wassermann reaction was markedly positive. The cerebrospinal fluid was normal in every respect. The patient was urged to undergo decompression craniotomy. A second oculist held the view that it was a case of typical optic neuritis. Antisyphilitic treatment was started. Two months later the pathologic changes in the optic nerve had retrogressed, but there was still slight decoloration of the papilla.

The second patient was a woman 45 years of age. She complained of headache and loss of vision. Examination revealed choked disk. The Wassermann reaction was markedly positive. The neurologic examination gave negative results. The initial diagnosis of a tumor of the brain was eliminated by the response to rigorous antisyphilitic treat

ment. The ocular symptoms vanished, and vision improved. The question arises what are the criteria arguing in favor of edema in a case of mechanical choked disk. The authors answer the question as follows: Ophthalmoscopic examination alone does not disclose much; the visual field must be examined. The initial symptoms of neuritis are intensive and rapid, subjective and functional. In the case of choked disk there is a disproportion between the pronounced objective ophthalmoscopic symptoms and the slight functional loss. In the case of choked disk the field of vision shows concentric regular contraction and coordination for white and all other colors. In the case of neuritis the narrowing is irregular and more pronounced with regard to colors. No chromatic disturbances are present in the case of choked disk, though they do exist with neuritis. The same thing holds good for disturbances in the adaptation to light. In the case of choked disk the cerebrospinal fluid is more apt to show pathologic changes. Neuritis may be cured by adequate treatment; on the other hand, improvement by means of decompression craniotomy would occur rarely in cases of choked disk.

The authors share Christensen's and Zamenhoff's opinion that the oculist must limit his examination to the fundus oculi, the field of vision and the acuteness of vision, leaving the diagnosis to the neurologist.

N. Blatt.

Retrobulbar Neuritis Among the Chinese: Report of Fifty-Three Cases. C. K. Lin, Chinese M. J. 50: 1345 (Oct.) 1936.

Retrobulbar neuritis is a disease involving the optic nerve, characterized by a selective affinity for the papillomacular fibers. Clinically it is represented by three cardinal signs: impaired vision, central scotoma and a fundus picture that shows disproportionally few or no abnormal findings to account for the loss of vision. The present knowledge of retrobulbar neuritis is still elementary. The etiology of the condition is obscure; of its pathologic features little is known, while its treatment is empirical and nonspecific in most cases.

Lin reports on the examination of fifty-three patients with this condition. The etiology was determined with a high degree of probability in twelve cases (22.6 per cent) and with some degree of probability in three cases (5.7 per cent); it remained unknown in thirty-eight cases (71.7 per cent). These figures are compared with the findings of Scheerer, Benedict, Gifford and Wilmer. The absence of multiple sclerosis and of tobacco and alcohol poisoning as causative factors in this series marked a striking difference from the findings in other countries. It is suggested that avitaminosis may play an important rôle in retrobulbar neuritis among the Chinese. The different forms of treatment are discussed; these include the administration of iodine compounds, fever therapy, sweat baths, the use of vitamins, the extraction of teeth and oral prophylaxis, and nonspecific therapy. With regard to the latter, as the condition is one with an obscure etiology and a transient course and as the patient is prone to recover spontaneously, the value of nonspecific therapy is extremely difficult to appraise. There is an extensive bibliography.   S. H. McKee.

Trachoma

THE RICKETTSIA OF TRACHOMA. H. FOLEY and L. PARROT, Compt. rend. Soc. de biol. 124: 230, 1937.

The presence of rickettsioid corpuscles in trachoma was confirmed. They are identified by the authors with inclusion "elementary" bodies. Trachoma is considered a local infection with a rickettsia, and the suggestion is made that the rickettsial conjunctivitis of sheep, swimming pool conjunctivitis and inclusion blenorrhea of the new-born belong to similar bacteriologic groups.

J. E. LEBENSOHN.

HISTOPATHOLOGIC CHANGES IN TRACHOMA AND IN OTHER FOLLICULAR DISEASES OF THE CONJUNCTIVA IN CONNECTION WITH THEIR PATHOGENESIS. A. J. POKROVSKY, Sovet. vestnik oftal. 9: 482 and 755, 1937.

This is a thorough study of the subject and is abstracted with difficulty. It occupies 84 pages and is subdivided into 22 chapters, with detailed discussion of various theories and of clinical and histopathologic data. Nineteen photomicrographs illustrate the articles. An abundant bibliography is appended. Pokrovsky comes to the following conclusions (condensed by the abstractor):

1. The conjunctiva of the human and of the animal eye is reconstructed and acquires adenoid qualities after birth. In its adenoid structure it resembles the mucous membrane of the respiratory and gastro-intestinal tracts and reacts similarly to irritation.

2. This reaction is observed mainly in the interfollicular lymphoid tissue.

3. The adenoid tissue, which is young and resembles the mesenchyme, in its reaction to irritation produces young cellular elements of lymphoid and reticulo-endothelial character.

4. The structure of the follicle of the subepithelial adenoid tissue is identical in all mucous membranes; it is unstable and may undergo identical phases of development.

5. The active phase of the embryonal center of the follicle is the result of irritation of the external medium. During this phase some new follicles form (physiologic irritation) and some die (pathologic irritation).

6. In follicular catarrh a change occurs in the details of the reaction which manifests itself in the number of follicles, in their location in relation to the epithelium, in their size, in the state of the embryonal center and in the degree of infiltration of the superficial layer of the conjunctiva.

7. In trachoma the details of the reaction are numerous and various. Outside of those mentioned, these are added: the degree of degeneration in the center of the follicle, the degree of infiltration of the interfollicular tissue and the degree of degeneration in the basal tissue of the conjunctiva.

8. However, proliferative changes with degeneration and frequent scar formation is the predominant picture in trachoma; by this it is differentiated from other conjunctival inflammatory processes.

9. The trachomatous process is not limited to the follicles. Pascheff's "laws of trachoma" are therefore one-sided and do not explain the basic process.

10. The trachomatous process is chiefly in the subepithelial adenoid layer of the conjunctiva. In severe forms the inflammation extends to the deep layers of the conjunctiva.

11. The study of the clinical material shows that the infiltrative forms predominate.

12. Neither the clinical nor the histopathologic data reveal the presence of a special hyperplastic (noninflammatory) process in trachoma.

13. The histopathologic and clinical study is of definite value, but bacteriology only will bring a final and decisive solution of the problem of the pathogenesis of trachoma.   O. SITCHEVSKA.

TRACHOMA IN CHINA. H. T. PI, Chinese M. J. **50**: 1465 (Oct.) 1936.

From writings in ancient classical books, trachoma must have existed in China at least since the time of the Emperor Huang Ti, in 2679 B. C., though no definite description of the disease has been found until 1752 A. D., in the Tang Dynasty. It was in the Ming Dynasty, in 1598 A. D., that Wang K'en Tang made accurate observations on this condition and described various clinical aspects of the disease.

It is found all over China, and at least one third of the whole population suffers from it. It is more prevalent in the north than in the south on account of the dry climate and dust storms in the northern section. Infection is spread chiefly by the wide use of common towels in public places and by the use of the same towels, wash basins and water in the household.

The complications and sequelae of trachoma are discussed. Particular emphasis is placed on the value of the early detection of mild pannus by means of the loupe and the slit lamp. The differential diagnosis of trachoma, folliculosis and vernal catarrh is discussed. The treatment of trachoma, especially the use of both surgical and medical treatment for the severe type of the disease, is outlined.
                                                          S. H. McKEE.

Uvea

CHOROIDAL TEARS. A. HAGEDOORN, Am. J. Ophth. **20**: 13 (Jan.) 1937.

Hagedoorn briefly discusses tears of the choroid and reports the case of a 17 year old girl who was struck in the right eye with a cardboard disk. Vision was normal. Under mydriasis a slightly curved streak was visible to the temporal side of the macula, a typical "rupture of the choroid." A diagnosis of rupture of the pigmented epithelium seemed more adequate but lacked an anatomic basis. Recently an eye enucleated for glaucoma demonstrated the possibility of such a lesion, and Tillema described such a rupture situated near the ora serrata.
                                                          W. S. REESE.

FAMILIAL HYALINE DYSTROPHY IN THE FUNDUS OCULI, OR DOYNE'S FAMILY HONEYCOMB "CHOROIDITIS." M. TREE, Brit. J. Ophth. **21**: 65 (Feb.) 1937.

Tree points out the fact that many pathologic processes of diverse origin in the macular region produce an ophthalmologic picture of whitish spots in the fundus and that a particular degeneration, namely hyaline change, may be the end-result of different diseases. To Doyne is credited the first definite dissociation of a familial type of "central choroiditis" as a distinct clinical entity. A brief description of Doyne's observations and Collins' histologic report on one of the eyes in Doyne's cases is given. The author reports on seven patients with Doyne's disease whom he has repeatedly examined, giving the refraction, the visual field, the light sense, the threshold for color, the appearance of the fundus and media and the results of physical examination. Among conditions that have sometimes given rise to confusion in diagnosis and which Tree describes are Tay's nonfamilial type of choroiditis, retinitis circinata, cerebromacular degeneration, retinitis punctata albescens, early mild disseminated choroiditis, arteriosclerotic retinitis, renal choroiditis and exudative choroiditis.

The article is illustrated.

W. ZENTMAYER.

Vitreous

WARMING OF THE VITREOUS IN THE RABBIT'S EYE BY SHORT WAVES. W. KOKOTT, Klin. Monatsbl. f. Augenh. **97**: 448 (Oct.) 1936.

In the first chapter of his paper Kokott discusses the technic applied and the results obtained with short waves by Schliephake, Liebesny, de Decker and Arendt, Sattler and others. Then he describes his own method, the thermic conditions of the rabbit's eye when short waves are used and the degrees of temperature obtained in the vitreous of the rabbit's eye by short waves. He arrived at the following conclusions: Single irradiations of from five to ten minutes are borne well by the animals, but untoward action may be observed on repeated raying in short intervals of time. The increase of temperature as such appears to have no unfavorable effect. Irradiation for more than ten minutes offers no advantages. The electrodes must be closely fitted to the head of the animal to prevent burns. It is essential to limit the therapeutically active field accurately so as to prevent undesirable effects on adjoining tissues. The question whether the results following short wave irradiation are actually produced by thermic action remains unsettled.

K. L. STOLL.

Sympathetic Ophthalmia

REPORT ON TWO CASES OF SYMPATHETIC OPHTHALMIA. C. SCHWARZENBURG, Chinese M. J. **50**: 1433 (Oct.) 1936.

This paper deals with two cases of severe sympathetic ophthalmia which was cured by injections of a solution containing cinchophen sodium, sodium salicylate and procaine hydrochloride.

The first case was one of sympathetic ophthalmia of short duration accompanied by neuritis and inflammation of the central and the

peripheral part of the choroid. In the course of eight months the patient received 112 injections of the aforementioned preparation and also 6 injections of a solution of sodium salicylate and caffeine sodium salicylate in methenamine. There was not a single general reaction, and acuity of vision improved from counting fingers at 0.5 meter to normal.

In the second case, acuity of vision was reduced to counting of fingers just in front of the eye. The patient was treated for two years and received 19 injections of cylotropin and 203 injections of atophanyl. These were well tolerated, and vision returned to normal.

S. H. McKee.

Therapeutics

PREVENTION OF OCULAR COMPLICATIONS IN TRYPARSIMIDE THERAPY.
M. FINE and H. BARKAN, Am. J. Ophth. 20: 45 (Jan.) 1937.

Fine and Barkan discuss the ocular dangers of tryparsamide therapy and note that in 95 per cent of the cases the unfavorable reaction occurs before the tenth injection. They therefore make a careful examination of the fields, fundi and vision before the first, third, fifth and tenth treatments and before every tenth treatment thereafter. Of 132 patients so treated, only 3 had unfavorable reactions, and these had not been properly checked as suggested here. Five other patients suffered impairment of the visual fields, which cleared up in from one to two months after cessation of the therapy.

W. S. REESE.

LOCAL TREATMENT OF OCULAR DISEASES BY RADIUM EMANATION.
G. LENZ, Klin. Monatsbl. f. Augenh. 97: 216 (Aug.) 1936.

Lenz discusses in detail the physical and therapeutic phases of treatment with radium emanation. Then he describes an apparatus which permits the dispensation of any medicamentous solution. This apparatus may be used for clinical and scientific purposes if combined with an electroscope. Lenz used oils and ointments for radium emanation for over three years in doses ranging from the smallest to those of from 100 to 300 electrostatic units. Among the ocular diseases treated were diseases of the lids, blepharoconjunctivitis, scrofular conditions of the conjunctiva and cornea and corneal scars, parenchymatous keratitis, trachoma and trachomatous pannus, and degenerative processes of the corneal epithelium and adjoining layers. This therapy requires further research, yet it has yielded results that warrant the following conclusions:

1. It is indicated in chronic inflammatory diseases of the epidermis of the lids.

2. It is indicated in nonpurulent inflammatory and degenerative corneal processes running a slow course, as soon as the height of the inflammation has passed. Regeneration and clearing of the tissues were greatly furthered by the emanation therapy. The causative factor was apparently not influenced by the treatment.

3. The treatment is contraindicated in yellow purulent infiltrative and ulcerous processes of the cornea, and in all inflammatory processes as long as severe irritation of the eyeball is present.   K. L. STOLL.

ULTRASHORT WAVE TREATMENT OF THE EYE. W. GUTSCH, Klin. Monatsbl. f. Augenh. **97**: 386 (Sept.) 1936.

Gutsch describes Schliephake's apparatus for the application of ultrashort waves to the eye and the essential points to be observed in its use. The apparatus has a wavelength of 6 microns and is fitted with glass electrodes. The main biologic and therapeutic response to these waves consists in immediate warming of the tissues, protracted hyperemia, cessation of pain, a marked afflux of leukocytes and increased phagocytosis in the inflamed tissues. Gutsch applied the rays from two to three times a week, beginning with an exposure of six minutes and gradually increasing the exposure to fifteen minutes.

No results were obtained in cases of corneal diseases, scleritis and glaucoma. The results were satisfactory in diseases of the fundus, ocular tuberculosis and paralyses of the ocular muscles and in purulent inflammatory processes of the lids, the tear sac and the orbit. Among those cured were seventy-eight persons with chronic iridocyclitis due to various causes or following operation; opacities of the vitreous cleared in some of them. Choroidal tubercles melted in two cases, leaving a smooth whitish scar on restitution of normal vision. Subjective improvement, which occurred sooner than usual, was noted in eighty cases of disease of the retina and choroid, in some of which the condition was acute, in others of the chronic degenerative type.   K. L. STOLL.

Society Transactions

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

March 15, 1937

JOHN H. DUNNINGTON, M.D., *Chairman*

LEGRAND H. HARDY, M.D., *Secretary*

SURGICAL INTERVENTION ON THE CORNEA. DR. RAMON CASTROVIEJO.

The cases in which corneal transplants were successful which were presented before the society proved that work on the problem of corneal surgical intervention has greatly advanced and from now on it must not be considered wholly experimental. There is a great deal still to be done to improve the results in difficult cases, and when a suitable technic is found the percentage of good results will compare favorably with that obtained with surgical intervention for retinal detachment and glaucoma.

ESSENTIAL PROGRESSIVE ATROPHY OF THE IRIS. DR. HUGH S. McKEOWN.

This article will be published in full in a later issue of the ARCHIVES.

RECOVERY FROM TOBACCO-ALCOHOL AMBLYOPIA WITH CONTINUANCE OF TOBACCO AND ALCOHOL. DR. FRANK D. CARROLL.

Alcoholic polyneuritis and the alcoholic type of pellagra are now considered to be deficiency diseases. Nutrition apparently plays an important rôle in tobacco-alcohol amblyopia. During the past two years a small series of patients with this condition were hospitalized, allowed to smoke and drink as much as they did while the amblyopia was developing and made to take a diet adequate in all respects but especially high in the vitamin B fraction. This was supplemented by brewers' yeast, vegex and wheat germ. Four sample cases in which this treatment proved successful were reviewed.

CHRONIC CATARRHAL CONJUNCTIVITIS PRODUCED BY STAPHYLOCOCCUS AUREUS. DR. PHILLIPS THYGESON.

The conjunctivitis-inducing power of staphylococci appears to depend on the production of an exotoxin. With potent toxins, conjunctivitis of varying severity can be produced in monkeys and rabbits. A close correlation between the production of toxin by staphylococci and their relationship to conjunctivitis in man is shown. The conclusion is reached

that toxin-forming staphylococci are an important cause of chronic catarrhal conjunctivitis.

·ORBITAL CYSTS WITHOUT EPITHELIAL LINING. DR. JOHN M. WHEELER.

This article will be published in full, with the discussion, in a later issue of the ARCHIVES.

April 19, 1937

JOHN H. DUNNINGTON, M.D., *Chairman*

LeGRAND H. HARDY, M.D., *Secretary*

CASE OF TUBEROUS SCLEROSIS. DR. BRUCE GROVES.

Tuberous sclerosis is a heredofamilial disease associated with mental deficiency and manifested by cerebral symptoms, Pringle's nodules or other fibromas of the skin, tumors of the viscera and brain and tumors of the retina or disk arising from the nerve fiber layer. These are found singly or in conjunction. The case of a man aged 43 with epilepsy and retinal tumor was reported as an example of this condition.

DISCUSSION

DR. DAVID WEXLER: Tuberous sclerosis and neurofibromatosis of Recklinghausen are closely allied, and symptoms of each may coexist in the same patient. In 1930 Dr. I. Goldstein and I described the case of a young woman with pigmented neurofibromas of the skin, a fibrosarcoma of the scapula and a distinctly low mentality. Unfortunately the fundi were not seen, owing to infiltration of the corneas. At autopsy careful examination failed to reveal lesions in the brain. Many melanotic nodules in the iris and melanosis of the choroid and sclera were observed. Since this report was published (ARCH. OPHTH. 3: 288 [March] 1930) these eyes have been carefully reviewed, and numerous small irregular flat and pedunculated growths were noted on the surface of the retina. They consisted of groups of fine, fibrillar, poorly differentiated glial tissue which pierced the membrane limitans interna. An occasional nucleus was seen.

INDUCED HYPERPYREXIA IN OPHTHALMOLOGY. DR. S. ARCHARD MORRIS.

A group of 16 persons with various ophthalmic conditions were treated with general physical hyperpyrexia in addition to the established local measures. Physical therapy appears to be of value in the ocular complications of gonorrhea, in iritis, in superficial punctate keratitis and in scleritis. It appears to offer little or no aid in trachoma, syphilitic uveitis and interstitial keratitis.

Recent Advances in the Bacteriology of Mooren's Ulcer. Dr. Isadore Givner.

A case of Mooren's ulcer was presented, in which there was a low phytotoxic index of Pels and Macht, suggesting a systemic toxin. Bacteriologic studies gave negative results. A demonstration of the gram-negative bacillus of Rodigina isolated from a patient with Mooren's ulcer in 1934 was given, together with the report of studies on its properties as shown on cultures and in experiments.

DISCUSSION

Dr. Rudolf Aebli: As Dr. Givner stated, the ulcer progressed after it was cauterized with trichloro-acetic acid. Then covered with a conjunctival flap, it seemed to be held in check for three or four weeks, after which it again began to spread. A Gifford delimiting keratotomy was then performed, and the wound was kept open daily for ten days. It is interesting to speculate about what cured the ulcer. Was the cure due to the production of an epithelial barrier, which shut off the advancing margin, or was it due to the production of prolonged hypotony?

Physiology of the Retinal Circulation. Dr. Fritz Bloch.

A new method of mapping the avascular area of the macula by means of Purkinje's figures produced by the retinal vessels was shown. With Bailliart's method the pressure in the retinal vessels of healthy persons was studied, and an average arterial pressure of 60 mm. of mercury systolic and 30 mm. diastolic was found. There was no measurable difference between the pressure in the right eye and that in the left eye. The capillary pressure was about 25 mm. of mercury, and the venous pressure nearly equaled the intra-ocular pressure.

Diseases of the Retinal Blood Vessels. Dr. Ervin Tusak.

The diseases of the retinal blood vessels were enumerated. The pathogenesis and pathologic changes in the tissue in blood vessels of various kinds and calibers were discussed. Emphasis was laid on the importance of arteriolosclerosis, an effect of hypertension. The diseases of distant organs which may affect the retinal blood vessels were reviewed.

DISCUSSION

Dr. S. A. Agatston: I wish to disagree with the speaker's statement about diabetes. He insists that diabetic retinitis is really caused by angiospastic disease, yet from my clinical studies I feel sure that is not so. One sees cases of advanced malignant hypertension in which the retinal vessels show no exudate and no hemorrhage, as Dr. Tusak himself showed. The arteries may be occluded and may be replaced by strands of connective tissue, yet there may be no hemorrhage and no exudative process. There are cases of essential hypertension in which the retinal vessels show marked signs of angiospasm and practically no hemorrhage. On the other hand, one sees the same picture of essential hypertension and the same arteriolar condition in a patient with hyperglycemia, and the hemorrhagic process is likely to appear. And one

may find diabetic patients with practically normal arteries and without hypertension, yet the patient may show a marked hemorrhagic process. I feel, therefore, that diabetes may cause retinitis because of a change in the composition of the blood, possibly due to a change in the osmotic pressure within the capillaries or to altered permeability of the capillaries. Furthermore, one sees hemorrhages in young diabetic patients who have no arteriosclerosis.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

March 18, 1937

CHARLES R. HEED, M.D., *Chairman*

ALEXANDER G. FEWELL, M.D., *Clerk*

LIGHT ADAPTATION AT THE MACULA: AN EXAMPLE OF ITS INDUSTRIAL IMPORTANCE. DR. EDMUND B. SPAETH.

This article will be published in full in a later issue of the ARCHIVES.

PATHOLOGIC CHANGES IN THE FILTRATION ANGLE IN SIMPLE GLAUCOMA. DR. PERCE DeLONG.

This brief paper reviews the pathologic processes and changes in the filtration angle.

THEORY AND PRACTICE OF TONOMETRY. DR. JONAS S. FRIEDENWALD.

Tonometric measurement is not a direct measurement of the intra-ocular pressure but is a measurement of the degree of indentation of the cornea produced by the tonometric plunger with a given load. As was noted by the earliest writers on tonometry, this measurement is not a record of the intra-ocular pressure alone, but is influenced by other factors, of which the distensibility of the eyeball as a whole is the most important, since indentation of the cornea with no net loss of the intra-ocular fluid must be associated with stretching of the ocular coats elsewhere. If one compares two eyes which have the same intra-ocular pressure but which differ markedly in the rigidity or distensibility of their coats, one will erroneously conclude from the tonometric reading that the pressure in the more rigid eye is higher than that in the more distensible eye. The present study represents an attempt to disentangle these two factors in the tonometric reading.

A study of the experimental data reported by others on the distensibility of the eyeball leads to the calculation of a simple mathematical relation between the volume and the pressure in any given eye, from which a coefficient of rigidity can be determined. If the eye could be distended by two different amounts and the pressure determined at these two different degrees of distention, the coefficient of rigidity of the eye could be determined. The studies which Schiötz undertook in calibrat-

ing his tonometer enable one to calculate the volume of corneal indentation corresponding to any given reading on the scale of the tonometer, i. e., the volume by which the eyeball is distended and the actual pressure in the eye carrying the tonometer. By making tonometric readings with two different weights one is able to determine the coefficient of rigidity of any eye and to arrive at a correct estimate of the intra-ocular pressure.

The results of this analysis indicate the following: 1. There is a systematic error in the Schiötz scale. The intra-ocular pressure is actually somewhat higher than the Schiötz scale indicates, a finding which is in agreement with the new comparisons of tonometric and manometric measurements that have been made on living eyes. (A corrected Schiötz scale for eyes of average rigidity is given.) 2. Clinically significant readings of the ocular rigidity can be made. 3. The rigidity of the eye increases with age, and its measurement is influenced by the axial refraction, the corneal curvature and certain drugs. In glaucoma, after a prolonged period of high tension the rigidity is abnormally high, but in glaucoma in which the tension is controlled the rigidity is normal. The rigidity of the eyeball is, therefore, an effect of glaucoma rather than a cause. In extreme myopia the rigidity of the eye is abnormally high, indicating that the coats of the eye have been stretched beyond their limit of elasticity.

DISCUSSION

DR. FRANCIS HEED ADLER: Did I understand Dr. Friedenwald to say that the smaller the eyeball the greater the rigidity of the coats, and that this is a general rule, with one exception, namely, in high myopia, in which the globe is excessively large yet has an extremely high degree of rigidity?

DR. ALFRED COWAN: Do you not think that the increased rigidity in very high myopia is probably due to the same cause as that found in uveitis?

DR. JONAS S. FRIEDENWALD: Since the relation of the change in the intra-ocular pressure to a measured change in the intra-ocular volume has been chosen as the coefficient of rigidity, it is to be expected that small eyes, though they may be perfectly normal, will show a higher coefficient of rigidity than large eyes. The same measured change in volume is proportionately greater in the case of the former than in that of the latter. For instance, the coefficient of rigidity, as determined from the experiments of Clark, previously referred to, is greater in monkeys than in cats and greater in cats than in dogs, as is to be expected from the difference in the size of the eyeballs of these different species. One would anticipate, therefore, that hyperopic eyes and eyes with a small radius of corneal curvature would have a higher coefficient of rigidity than myopic eyes and eyes with a large radius of corneal curvature. This relationship was found to hold as predicted, except in the case of extreme myopia, in which the distensibility of the eyeball is less than the large size of the eyeball would lead one to predict. The cause of this relative increase in the rigidity of the eyeball in extreme myopia has not been ascertained. I have suggested a possibility that the sclera in these cases has been stretched beyond its elastic limit, but other explanations may be advanced which are at least equally reasonable.

Book Reviews

Tenth Annual Report of the Giza Memorial Ophthalmic Laboratory, Cairo, 1935. Price, 25 piasters. Pp. 136, with illustrations. Cairo: Schindler's Press, 1936.

The tenth annual report records the administration and scientific work of the laboratory during 1935 under the following four headings:

I. *Postgraduate Education.*—The usual postgraduate course in medical and surgical ophthalmology was given during April and October. Twenty candidates for admission to the ophthalmic section of the department of public health attended the course given in April and fourteen the course given in May.

II. *Pathologic Section.*—Five hundred and twenty-eight pathologic specimens and 16,064 smears from the eyes of patients with purulent ophthalmia were submitted for examination. Among the 139 blind eyes received, the sight in 89, or 64 per cent, had been destroyed through acute ophthalmia. Among the interesting specimens studied were 1 showing xeroderma pigmentosum, 2 showing uveal tuberculosis, 1 showing involvement of the eyelid with an infection due to filaria Bancrofti, 2 showing neurofibroma and 1 showing a true neuroma believed to have arisen from the sympathetic ganglions of the lacrimal gland. There were 5 cases of "fly-blown orbit" due to infestation with the eggs or larvae of Wohlfahrtia magnifica.

The statement is made that from 60 to 70 per cent of epithelial scrapings from the conjunctiva of patients attending the Government Ophthalmic Hospital, Giza, show eosinophils or eosinophilic granules. The presence of a fine tenacious pellucid membrane which can be stripped from the conjunctiva is considered to be of much greater diagnostic value in spring catarrh than eosinophilia.

III. *Clinical Section.*—Among the interesting conditions observed at the laboratory during the year were gangrene of the left upper eyelid; tuberculous abscess of the cornea; subconjunctival dislocation of the lens; a peculiar form of cataract associated with glaucoma; asteroid hyalitis, and familial cerebromacular degeneration. There are short descriptions of the clinical features of each case reported and many representative photographs and colored drawings.

IV. *A. Pathologic and Bacteriologic Research.*—The etiology of trachoma is still the major research problem of the laboratory, but no account of the work in progress for 1935 is included in the report. Dr. R. P. Wilson, the director of the laboratory, has continued his study of the seasonal incidence of acute ophthalmia and has made observations in different parts of Egypt under varying conditions of temperature and humidity. In Egypt the principal causes of infectious conjunctivitis are the gonococcus, the Koch-Weeks bacillus and the diplobacillus of Morax and Axenfeld. The diplobacillus appears to have no true seasonal variation, but Koch-Weeks conjunctivitis and gonococcic conjunctivitis habitually become epidemic during the spring and fall.

A study of the twenty-seven specimens showing sympathetic ophthalmia received by the laboratory during the past fifteen years was made by Dr. Ibrahim A. Mohamed, the assistant pathologist. The outstanding findings of the study were: (1) the high proportion of cases in which an operation was the original trauma and (2) the large number of cases in which there was no trauma.

B. Clinical and Therapeutic Research.—The experimental work carried out during the past few years at the village of Bahtim, near Cairo, was continued. It was found impossible to prevent trachoma and acute ophthalmia by means of prophylactic drops, such as zinc sulfate, acriflavine hydrochloride and silver nitrate.

Therapeutic tests were made with a number of proprietary preparations submitted for use in trachoma. None was found to have an advantage over copper sulfate and crude chaulmoogra oil, the remedies commonly employed. A fever-producing preparation was found to compare favorably with milk in the treatment of gonorrheal ophthalmia.

The annual report closes with two appendixes, "The Various Forms of Conjunctivitis Among Children in Egypt and the Near East," by R. P. Wilson, and "Tebeprotin in Diagnosis and Treatment," by Ibrahim A. Mohamed.   PHILLIPS THYGESON.

Atlas der Augenkrankheiten. By Prof. Rudolf Thiel, Frankfort-on-Main. Price, 24 marks. Pp. 196, with 420 illustrations, mostly in color. Leipzig: Georg Thieme, 1937.

Starting with the premise that one remembers what one sees better than what one hears, the author has written a textbook on ophthalmology which consists mainly of illustrations with a brief explanatory text. In the text will be found a description of the important symptoms, etiology and treatment of diseases of the eye. The arrangement of the subject matter is not the usual one but is the one easiest to follow in teaching; thus, the anatomic and pathologic features and the operative treatment, if any is indicated, are given for each clinical condition. The illustrations are principally photographs, but drawings are used when details are necessary. The pictures of the fundus are drawings in color.

A study of the book quickly shows that it admirably fulfils its purpose, and the author is to be congratulated. The illustrations are excellent, and those in color, particularly, show up well. The student or practitioner will find a vast number of conditions admirably described. A possible adverse criticism might be that the text is too brief, particularly as regards the outline of treatment and operative procedure, but it is to be remembered that this is primarily an atlas and hence is to be used only in conjunction with a textbook. Finally, mention should be made of the remarkable reasonableness of the price of the book; here is an atlas with four hundred and twenty illustrations, mostly in color, which sells for 24 marks.   ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66, Boulevard Saint-Michel, Paris (6e).
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

INTERNATIONAL OPHTHALMOLOGIC CONGRESS

Secretary: Dr. E. Marx, Costzeedijk 316 Rotterdam, Holland.
Place: Cairo. Time: Dec. 8-14, 1937.

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

President: Dr. A. F. MacCallan, 33, Welbeck St., London, W., England.

FOREIGN

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

GERMAN OPHTHALMOLOGICAL SOCIETY

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. T. E. Ashdown Carr, 34, Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3, Midan Soliman Pasha, Cairo.
Time: March 1938.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Time: July 8-10, 1937.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.

Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.

Place: Lindley'a 4, Warszawa.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.

Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. F. Berg, Uppsala, Sweden.

Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.

Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.

Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.

Place: San Francisco. Time: June 13-17, 1938.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Lee W. Dean, Washington University Medical School, St. Louis.

Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

American Ophthalmological Society

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.

Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.

Place: Hot Springs, Va.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.

Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.

Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

New England Ophthalmological Society

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time:
8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City, Utah.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Salt Lake City, Utah. Time: May 24-27, 1937.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. Frank Brodrick, 302 First Ave., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, Swedish-American Bank Bldg.,
Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each
month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each
month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Secretary: Dr. O. M. Marchman, Medical Arts Bldg., Dallas, Texas.

Southwestern Michigan Triological Society

President: Dr. O. B. McGillicuddy, 1908 Capitol Band Tower, Lansing, Mich.
Secretary-Treasurer: Dr. Maurice C. Loree, 120 W. Hillsdale St., Lansing, Mich.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. C. W. Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all
members have served.
Secretary: Dr. Edna M. Reynolds, 227 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the
month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Bldg., Atlanta, Ga.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.
Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York City.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. Rosenberger, 221 Fifth St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514 Sixth St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary-Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Edwin W. Burton, University of Virginia, University.
Secretary-Treasurer: Dr. George G. Hankins, 202 Medical Arts Bldg., Newport News.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. Samuel T. Hubbard, 294 State St., Hackensack, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Thurber LeWin, 112 Linwood Ave., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. G. Henry Mundt, 30 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Andrew Timberman, 21 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Claude S. Perry, 40 S. Third St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. A. W. Davidson, City National Bank Bldg., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 720 Medical-Professional Bldg., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

LONG BEACH, EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Hychener, 130 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LeGrand H. Hardy, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

PITTSBURGH SLIT LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third
Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and
scientific meeting 6:30 p. m., fourth Friday of each month from October to
April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio,
Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each
month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Fourth
Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except
July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Philip B. Green, Old National Bank Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of
each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. J. Werfelman Jr., 725 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse,
N. Y.
Place: University Club. Time: First Tuesday of each month except June, July
and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto.
Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. James M. Greear Jr., 1740 M St., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Ernest Sheppard, 927 17th St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday
in November, January, March and May.

ATROPHY OF THE OPTIC NERVE AND NAEVUS FLAMMEUS ASSOCIATED WITH HEMANGIOMA OF THE CHOROID

REPORT OF A CASE

PHILIP JAMESON EVANS

BIRMINGHAM, ENGLAND

The following case is reported because of the possible relation of the condition to encephalotrigeminal angiomatosis, which was recently reviewed by Appelmans.[1]

REPORT OF CASE

History.—A. C., a boy aged 12 years, came to the Birmingham and Midland Eye Hospital on Dec. 30, 1935, complaining of pain in the left eye of three weeks' duration. The left eye had been blind since birth, and on occasions it had been painful for short periods.

Examination—The condition noted on examination was as follows:

Right Eye: Vision was 6/6. The eye was structurally and functionally normal. There was no evidence of abnormal vessels. The tension was 17 mm. of mercury (Schiötz). The field of vision was full as determined by perimetry with the use of a 0.5 degree red test object and a 0.5 and a 0.25 degree white test object at 0.3 meter.

Left Eye: Vision was reduced so that there was no perception of light. There was a deep zone of dilated ciliary blood vessels forming a prominent ciliary flush. In addition, the conjunctival vessels were dilated. The cornea was diffusely edematous: the anterior chamber was shallow and in its lower part presented a small hyphema. The vessels of the iris were prominent, and several large vessels were observed coursing irregularly on the anterior surface. The lens was intumescent and opaque. The tension was 42 mm. of mercury (Schiötz). Transillumination gave an even reflex, but this was somewhat more diminished than the opacity of the lens would lead one to expect.

Face: The face presented the condition of naevus flammeus limited strictly to the left side of the midline; the extent of the distribution is best seen in figure 1; briefly, it involved chiefly the distribution of the maxillary division of the trigeminal nerve, with some overlapping in the area of the ophthalmic division. The mucous membranes of the left cheek, the left side of the upper jaw and the hard and soft palate were involved.

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1. Appelmans, M.: Encephalotrigeminal Angiomatosis, Arch. d'opht. **52:**835 (Dec.) 1935.

Body: The body showed numerous areas of hemangiomatosis. A complete neurologic examination was carried out, but no lesions involving the nervous system were discovered.

Roentgenograms of the Skull: There was no visible area of calcification in any part of the skull. The left optic foramen was slightly larger than the right.

Enucleation.—On the basis of the history of the recent incidence of the attack of pain in the left eye present at the time of admission, and on the basis of the result of examination on transillumination, it was felt that in all probability the present state of the eye was due to hemorrhage from a choroidal hemangioma. Excision of the eye was advised and was performed, without unusual bleeding, on December 31, with the patient under general anesthesia. Recovery was uncomplicated, and healing of the socket was normal.

Pathologic Examination.—The enucleated eye was subjected to pathologic examination. The report from the department of pathology is as follows: "Sec-
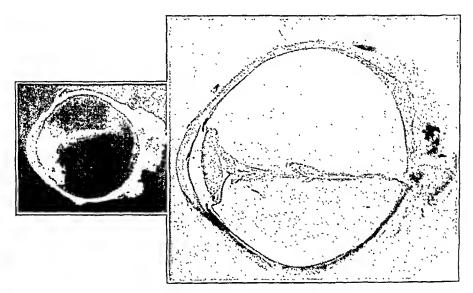
Fig. 2.—Hemisections of the left eye, showing the opaque lens pushed forward, the retina folded up into a central cord of tissue and completely detached and the flat hemangioma of the choroid occupying the posterior part of the globe.

tion shows keratitis with vascularization of the cornea; anterior synechiae; degenerated lens; detached retina; large angioma of choroid, and cupped disk" (fig. 2).

The microscopic study of the sections showed a number of points:

1. The filtration angle of the anterior chamber was completely closed by the forward projection of the lens and iris (fig. 3).

2. The optic disk was deeply cupped, and the retina was seen to be attached to its margin (fig. 4).

3. The optic nerve was diffusely infiltrated by small hemorrhages (fig. 5).

4. The retina, which was greatly degenerated, had been completely detached, except at the margin of the disk, and was seen as a double fold passing forward in the center of the globe. The space around was, in the fresh state, occupied by subretinal fluid and hemorrhage.

Fig. 1.—Colored photograph of the patient.

Fig. 3.—Photomicrograph of a section of the filtration angle.
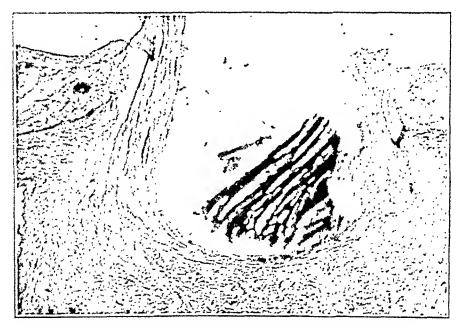
Fig. 4.—Photomicrograph of a section of the optic disk.
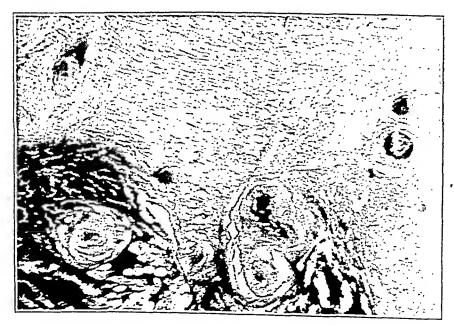
Fig. 5.—Photomicrograph showing small hemorrhages in the optic nerve.
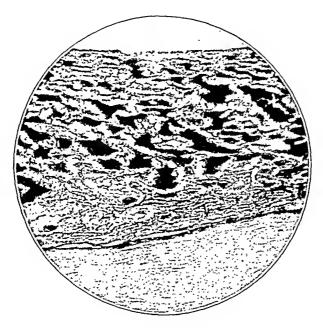
Fig. 6.—Photomicrograph of a section of the hemangioma of the choroid.
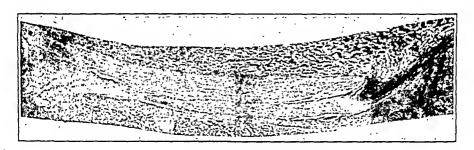
Fig. 7.—Photomicrograph showing the path of the neoplastic tissue.

Fig. 8.—Photomicrograph of a section of episcleral tissue, showing the presence of dilated vessels.

5. In one hemisection of the globe a circular, ill defined mass was seen projecting slightly from the surface of the choroid; its color was reddish brown. Portions of this tissue, on section, proved to consist solely of blood clot.

6. The posterior half of the choroid was seen to be greatly thickened, the widest part corresponding with the posterior pole; on section this thickening was seen to be composed of a diffuse hemangioma (fig. 6).

7. The lens was cataractous and swollen, but the changes were of recent origin.

8. Serial sections of the globe showed the path of entry of the neoplastic tissue. This followed the course of one of the venae vorticosae. Its path from the growth of the choroid, in the region just behind the equator of the globe, could be seen passing obliquely backward through the sclera and thence outside the globe to one of the greatly dilated vessels lying to the side of the optic nerve (fig. 7).

9. The episcleral tissue also showed the presence of many very dilated vessels which appeared to have the structure of veins (fig. 8).

COMMENT

From the history of attacks of pain in this case it seems probable that hemorrhages from the choroidal hemangioma, with rise of tension, had occurred previously from time to time. The advanced condition of degeneration of the retina and the completeness of its detachment lend color to this supposition. Consequently, one is not in a position to confirm the findings of Thiel that cupping of the optic disk and atrophy of the optic nerve are independent of tension. On the other hand, the fact that the patient went for long periods without pain in the eye and that the intumescence of the lens was of recent development suggest that, apart from the few occasions mentioned, the tension in the eye was not raised, and it is extremely probable that so advanced a degree of cupping is the result of essential atrophy of the optic nerve, consequent on defective vascular nutrition, and is not the result of prolonged increase of the intra-ocular pressure.

THE JAW-WINKING PHENOMENON

REPORT OF A CASE

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DETROIT

It is with some hesitation that I report a case of the jaw-winking phenomenon, since I know full well that it has little clinical value and that the condition is so rare as to be possibly unimportant. However, it is' felt that the rarity and curiousness of the disorder are in themselves sufficient reason for bringing it to the attention of ophthalmologists.

The condition which has been called, rather inelegantly, the jaw-winking phenomenon was first described by Marcus Gunn in 1883, when he showed a patient with this disorder before the Ophthalmological Society of the United Kingdom. Since that time about ninety additional cases have been reported, although there is some reason to question the authenticity of some of them. In 1895 Sinclair [1] collected a number of instances and discussed several possibilities as to the etiology. Sym [2] in 1908 reported a case and again reviewed the etiologic considerations. The review which I made of the literature does not pretend to be complete, but it is hoped that a summary of a few of the more typical cases, together with a description of the present case, will serve to affirm the opinion that the phenomenon is a definite clinical entity.

REPORT OF CASE

A woman of 41 years presented herself, complaining of difficulty with near work and considerable lacrimation. She showed a moderate amount of compound hyperopic astigmatism in each eye and slight presbyopia, for which glasses were prescribed. The rest of the examination gave entirely negative results except for a very small amount of ptosis of the upper lid of the right eye. This was manifest only when the eyes assumed a position of rest, looking straight ahead (fig. 1 A). When the eyes looked upward the eyelid readily retracted, showing no paralysis. The ocular movements were unlimited in all directions. There was no evidence of Horner's syndrome to which the ptosis could be attributed. With a red glass no diplopia could be elicited. The pupils reacted directly and consensually to

1. Sinclair, Walter W.: Abnormal Associated Movement of the Eyelids, Ophth. Rev. **14**:307, 1895.

2. Sym, William George: A Case of Partial Ptosis with Exaggerated Involuntary Movement of the Affected Eyelid: The "Jaw-Winking" Phenomenon, Ophth. Rev. **27**:197, 1908.

light and normally in accommodation. The patient stated that the ptosis had existed for as long as she could remember. She stated also that whenever she ate or chewed gum vigorously the upper lid of the affected eye "winked," that is, the upper lid moved up and down rapidly. This curious retraction of the upper lid was evidenced when she merely moved the lower jaw downward and was much more marked when she moved the lower jaw to the left (fig. 1 B). Movement of the jaw caused no winking or jerking of the left eyelid. The condition caused the patient little concern, since it was apparent only when she executed rather vigorous movements of the lower jaw. There was no history of a similar condition in her immediate family, but she had been told when she was a little girl that she had an eye "just like" her aunt's.

Fig. 1 (the author's case).—A, the aspect at rest; B, the aspect on movement of the jaw to the left.
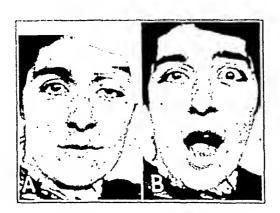
Fig. 2 (case of Sym [1908]).—A, the aspect at rest; B, the aspect during sudden depression of the jaw.

CASES REPORTED IN THE LITERATURE

In 1908 Sym reported the case of a woman of 30 years. The only fault which he found with the eyes was that which is well illustrated by figure 2. There was a distinct degree of ptosis of the left upper eyelid (fig. 2 A). The levator muscle was not paralyzed, as was shown by the fact that the patient could elevate the lid readily on looking upward. Movements of the globe were perfect. When, however, the patient was chewing her food or when she was singing, the upper lid might almost be said to spring upward. When she suddenly opened her mouth by depressing the lower jaw, the lid sprang up in the manner shown in figure 2 B. When the lid flew up there were no

corresponding movement of the right upper lid, no corrugation of the forehead and no untoward movement of the eye itself. There was no evidence of Horner's syndrome.

In 1912 Thomson and Souter[3] reported a case of this condition in a girl of 9 years (fig. 3 *A*). When the eyes were at rest and looking forward the patient showed a slight degree of ptosis of the left upper eyelid. She showed no miosis and no evidence of unilateral sweating or flushing. The ocular movements were normal in all directions. When the mouth was opened slowly there was a very faint upward jerk of the left upper lid, but when the mouth was opened rapidly and worked to the right there was a marked upward jerk of the lid (fig. 3 *B*) and 2.2 mm. of the sclera was exposed above. When the jaw was worked to the left there was no movement of either upper lid. The patient was unable, while looking forward in an ordinary way, to raise either eyelid without using the frontalis muscle or opening her mouth, but the lids became elevated equally with upward move-

Fig. 3 (case of Thomson and Souter [1912]).—*A*, the aspect at rest; *B*, the aspect on movement of the jaw to the right.

ments of the eyes. The lids closed equally well, both in sleep and on forced closure. General physical examination, both of this patient and of the one previously mentioned, revealed nothing significant.

Wilkinson[4] and Coburn[5] each reported a case of similar elevation of an upper lid associated with movement of the lower jaw. Coburn's patient was unable to open his left eye, the one affected, except when he opened his mouth. Wilkinson's patient was able to open and close the eyes normally, except that when he made an extra effort the right, or affected eye, opened a little wider. In a case reported by Lutz[6] in

3. Thomson, J. E. G., and Souter, W. C.: Congenital Ptosis with Associated Lid Movements of the Affected Eye, Ophth. Rev. **31**:172, 1912.

4. Wilkinson, cited by Wood, Casey A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1913.

5. Coburn, cited by Wood, Casey A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1913.

6. Lutz, Anton: Jaw-Winking Phenomenon and Its Explanation, Arch. Ophth. **48**:144 (March) 1919.

1919 the phenomenon in question was first noticed at the age of 5 years. There was a slight ptosis on the right side, and with a colored glass diplopia could be elicited, corresponding to that caused by paresis of the right superior oblique muscle. Elevation of the right upper eyelid could be induced by opening the mouth or by moving the jaw from side to side, but the patient could also elicit the movement voluntarily, without any movement of the jaw. The latter finding is a peculiarity which was not present in any of the other reported cases. It is impossible normally to elevate one lid alone.

In Holloway's [7] case of a woman aged 44 there was a history of distinct drooping and retraction of the right lid at times, or, as the patient expressed it, she winked while she ate. The fields showed diplopia and indicated paresis of the right superior rectus muscle. The eyes were otherwise normal. Two additional cases were reported, one by Kleinhans and one by Menacho. In both these patients, who had ptosis of one eyelid, the lid shot upward when the mouth was opened, but lateral movements of the jaw had no such effect.

In the reported cases the error was about equally distributed between the right and the left side. No case was reported in which both sides were involved. Males are affected about as often as females. In most cases ptosis was present on the affected side, although a few cases were reported in which no ptosis was evident.

Several explanations have been offered as to the probable meaning of these associated movements. After Gunn made the first report of a case of this condition a committee was appointed by the Ophthalmological Society of the United Kingdom to consider it. The explanation offered by this committee is by far the most satisfactory submitted, namely, that the levator muscle receives motor impulses from both the third and fifth nerve. Sym concluded his paper on the subject as follows:

There does not appear to be any other probable explanation of this curious association than that in some inexplicable way there arises some confusion in the joining up of fibers and cells belonging to the fifth and third nuclei in such a fashion that the levator receives less than its normal innervation, and there is, therefore, a certain degree of ptosis, but without any paralysis of the muscle, which is capable of full contraction. At the same time the levator receives some fibres which are "intended for" one or more of the muscles of mastication and when these muscles are put in action, at all events when put strongly in action, the levator is unintentionally innervated, producing the curious effect described.

This explanation is anatomically conceivable, since the nuclei of the third and the fifth nerve closely approximate each other as they lie

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7. Holloway, cited by Wood, Casey A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1913.

in the floor of the sylvian aqueduct, and they are connected by the posterior longitudinal fasciculus. That the lesion is either nuclear or very close to the nuclei is shown by several cases in which the patient showed paresis of one or more ocular muscles. Lutz agreed that the lesion was probably supranuclear and very close to the nuclei but expressed the belief that it had to do with the inhibitive mechanism of the levator. He has published an excellent review of the various theories. Menacho observed that in his case and in one or two other reported cases, the condition appeared to be acquired rather than congenital, and he stated the opinion that an exclusively anatomic explanation was unjustified. He recognized the close relationship which exists between the functions of the cranial nerves and argued that the condition was due to an alteration of this functional relationship rather than to an anatomic alteration. When a review of the published cases is made, however, it is noticed that in the majority the condition is described as congenital and in the remaining few it is said that the condition "was not noticed" until some time after birth. The fact that the condition was not noticed does not disprove its existence.

Bishop Harman attempted to show that the condition is an atavistic anomaly. He thought it was merely a manifestation of a reversion to a primitive type, such as, specifically, the shark, in which the cranial nerves are more closely associated than in man. His conclusions are not, it must be confessed, very convincing.

Ole Bull considered that such a movement is purely a reflex and what is constantly seen in children with blepharospasm who open their mouth when they wish to open their eyes. This theory can be objected to on the ground that in all cases only one eye is affected and that in some cases the associated movement of the lid is elicited only on lateral movement of the jaw.

There have been cases reported under the name "jaw-winking" which are not true instances of the condition. Schirmer reported the case of a man aged 28 in whom facial paralysis on the left had developed. Four months later he began to show slow improvement. After three more months he was unable to wink his left eye, although the nerve supply to the other facial muscles showed considerable evidence of recovery. It was noted, however, that movements of the left cheek, in laughing or chewing, were accompanied by partial closure of the left eye. The reporter felt that these associated movements were due to the fact that fibers from the central stump of the facial nerve that were intended for the cheek muscles had grown into the orbicularis muscle. There was no involvement in this instance of the muscles used in mastication or of the levator palpebrae superioris, so the case cannot be considered a true case of jaw winking.

A similar case was reported in January 1937 by Temple Fay[8] The patient was a woman aged 42 who had had a transplantation of the facial nerve on the right. She had previously had facial paralysis on the right after operation on the mastoid. After sixteen months the facial nerve on the right showed some evidence of regeneration but with a rather bizarre result. Dr. Fay stated:

When the command to close the right eye was given, not only did the right corner of the mouth draw up, but even with the wink reflex on the left, there was a definite jerk or twitching at the lower angle of the mouth on the right. . . . When the patient forcefully closed the eye, the effort gave a response in the lower ⅔ of the face. When instructed to draw up the corner of the mouth, the patient showed a slight twitching movement beneath the right eye.

In this case, as in the one previously cited, neither the muscles used in mastication nor the levator muscle was involved, and the case cannot be considered a true case of jaw winking.

In none of the articles reviewed was any mention made of treatment, and in many of the cases, including the one reported by me, treatment was neither desired nor advisable. It might not be out of place to suggest, however, that in those cases in which ptosis is pronounced and in which there is no paresis of the superior rectus muscle a tenotomy of the levator muscle might be done, to be followed later by transplantation of one third of the tendon of the superior rectus muscle to the upper lid (Motais' operation). Such a procedure would produce a satisfactory cosmetic result and at the same time provide for functional action of the upper lid.

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8. Fay, Temple: Jaw-Winking, Arch. Neurol. & Psychiat. **37**:208 (Jan.) 1937.

LOCALIZATION OF INTRA-ORBITAL FOREIGN BODIES

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PHILADELPHIA

PRINCIPLES OF ROENTGEN TECHNIC

The localization of intra-ocular foreign bodies is a rather special department within the general field of roentgenology and involves many problems which are of great importance to both the ophthalmologist and the roentgenologist. The ophthalmologist is far too prone to accept without question the indicated position of a foreign body on a chart showing the localization which has been handed to him from the roentgenologic department. This chart represents as accurately as possible within the scope of the method used and the experience of the roentgenologist the position of the foreign body in relation to the ocular structures. There are, however, so many possible errors that it is to be highly recommended that the ophthalmologist thoroughly familiarize himself with the principles involved and the methods used, so that he may follow the procedure intelligently and not accept the data on the localization chart at face value.

There is no problem in medicine requiring mutual cooperation to a greater degree than that of the localization of an intra-orbital foreign body. It would be a matter of relative simplicity were the globe visualized on the x-ray film, but such is not the case, and as there are many bony structures superimposed over the area of the globe the problem becomes complex.

It is important for the ophthalmologist to have definite data on the following points in regard to an intra-orbital foreign body: (*a*) whether it is visible on roentgen examination; (*b*) its approximate size; (*c*) its relation to the structures of the globe; (*d*) whether it is intra-ocular or extra-ocular.

The fact that numerous methods and apparatus are used for localization, some workers relying on one method and others on a different set of principles, shows clearly that no single procedure can be relied

4. *Roentgenographic Stereoscopic Methods.*—Several procedures have been recommended, one of which was reported by Béclère and Morax [5] in 1907. We found these to be less accurate than the other standard methods because of the narrow angulations involved and because they require considerably more experience for the same degree of accuracy to be attained.

5. *Accessory Methods.*—These include: visualization of the anterior portion of the globe free from interference by the bony structures; the injection of air or opaque material into the capsule of Tenon to identify the posterior part of the sclera; autovisualization, in which the patient identifies and checks the location of the foreign body by the sensitivity of his retina to the roentgen rays, and the methods in which various markers are used which have been previously introduced in the conjunctival sac or sewn to the lids to assist in identifying the plane of the cornea.

Comment.—All the principles and methods just described should be thoroughly known by the radiologist who attempts to assume responsibility in reporting on cases of an intra-ocular foreign body. This does not mean that he must be familiar with every device, but if one method leaves doubt in the mind of the ophthalmologist the roentgenologist should be able to clear this up by using the principles of whatever other procedure is required to settle the problem at hand.

The underlying geometric principles as applied to any of the methods already described are of supreme importance, and failure to observe these means inaccurate work. Therefore, the ophthalmologist will find an understanding and application of the following simple geometric principles of the greatest value in checking the roentgenographic procedure.

Principle 1 : A false impression is given as to the size of a foreign body owing to magnified distortion (fig. 1 A). Because of the spreading of the roentgen rays, which obey the law that the intensity of light is inversely proportional to the square of its distance from the source, the resulting image is magnified.

Principle 2: A false impression is given as to the size of the foreign body owing to true distortion, if its axis is not parallel to the plane of the x-ray film (fig. 1 B and C). The actual working conditions with a modification of the localizer of Sweet are represented in figure 1 D, from which one can readily see that the measurement and the size of the foreign body on the x-ray film may be misleading.

5. Béclère and Morax: Un nouveau procédé de localisation des corps étrangers métalliques intra-oculaires par la stéréo-radiographie avec repères cornéens, Rev. gén. d'opht., 1907.

on for accuracy in all cases. The roentgenologist is often too prone to accept whatever practice he has been taught in his period of training or is prevalent in his community. The ophthalmologist is also likely to accept without question the results obtained on a diagrammatic chart. My associates and I do not recommend adherence to any single procedure of localization exclusively but believe that if both these specialists understand and consult together regarding the problems presented by their cases the accuracy of the results will be greatly improved. The underlying principles of the roentgen rays as applied to this special problem are similar to the general principles of roentgenography, but assume a far greater importance from a geometric standpoint because of the extreme accuracy required. I shall therefore briefly point out the methods and factors entering into this problem which I believe would assist the ophthalmologist in his interpretation of the result.

ROENTGENOGRAPHIC METHODS

1. *Simple Method.*—This consists of two roentgen projections at right angles to each other, from the postero-anterior and the lateral aspect. This merely identifies the presence of the foreign body and shows its general relationship with the bony structures of the orbit. At the present day this is recommended only as a means of identification. Such a method of localization was the first reported in the literature, having been described by Clark [1] in 1896.

2. *Geometric Methods.*—These localize the position of the foreign body in relation to some fixed marker outside of the globe, which is placed in a known relation to the plane of the cornea. These methods are the most commonly accepted, and most of them are sufficiently accurate for ordinary work. The first of these methods to be reported was described by Sweet [2] in 1898.

3. *Physiologic Methods.*—These depend on the rotation of the globe between predetermined points of visual fixation and the resulting behavior of the foreign body with regard to the optic center of the eye. An example of this type of method was reported by L. Webster Fox [3] in 1902 and another by Köhler [4] in 1903.

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1. Clark, C. F.: A Question as to the Presence and Location of Minute Fragment of Steel in Eye Determined by the Roentgen Rays, Tr. Am. Ophth. Soc. **7**:711, 1894-1896.

2. Sweet, W. M.: The Roentgen-Rays in Ophthalmic Surgery, Am. J. M. Sc. **116**:190, 1898.

3. Fox, L. W.: The Localization of Foreign Bodies in the Eye, Philadelphia M. J., 1902; Lancet, 1902.

4. Köhler, A.: Zur Technik des Fremdkörpernachweises im Augapfel, Fortschr. a. d. Geb. d. Röntgenstrahlen **6**:190, 1903.
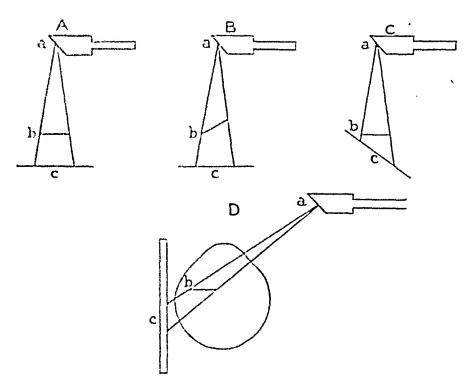
Fig. 1.—A, magnified distortion due to spreading of the beam of the roentgen rays. B, true distortion due to the angle of the foreign body. C, true distortion due to the inclination of the surface of the x-ray film. D, relationship of the x-ray tube, the eyeball and the film in the modified method of localization of Sweet. The letters have the following significance: a, the target of the x-ray tube; b, the foreign body, and c, the x-ray film.
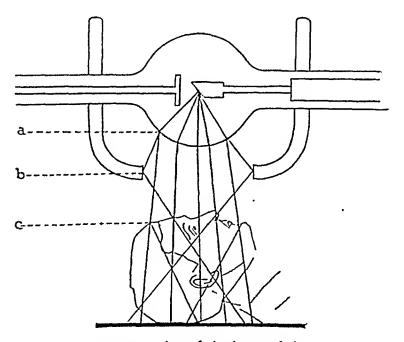
Fig. 2.—Diagram showing scattering of the beam of the roentgen rays wherever it strikes against a hard surface. The main points from which this scattering occurs are: the glass bulb of the x-ray tube (a), the lead glass or metallic container (b), and the tissues of the patient's head (c).

These factors of distortion are minimized by putting the greatest practicable distance between the x-ray tube and the surface of the film, which makes for more nearly parallel beams of radiation, and also by the approximation of the injured eye as near as possible to the surface of the film, which gives a sharper image on the resulting roentgenogram.

Axiom 1. In all cases of localization of a foreign body the apparatus should be so constructed as to have the greatest practicable distance between the tube and the film and the smallest possible distance between the injured eye and the film.

Principle 3: Definition or sharpness of outline on the roentgenogram depends on the minimum of distortion, the smallest possible degree of scattered radiation, perfect immobilization of both the head and the eye and a fine effective focal spot in the target of the x-ray tube.

Scattered rays tend to blur the outline. The scattered rays are generated from the x-ray tube, the walls of the holder and the head of the patient (fig. 2). This scattering is avoided by using the smallest possible opening for the emission of the roentgen rays.

If one were dealing with a point source from which the roentgen rays were emitted, one would have to deal only with distortion, scattering and movement, but as the focal spot of the roentgen rays is of a measurable diameter the foreign body is often smaller than the source of the rays. This brings an important factor into the problem. The resulting phenomenon is similar to that of the heavenly bodies. In an eclipse of the sun, in which the moon passes between the sun and the earth, the moon casts a shadow on the surface of the earth in which the central portion, or the zone of total eclipse, is denser than the periphery, or the zone of partial eclipse. The area of total eclipse is the umbra, the peripheral zone being the penumbra. A similar phenomenon is present in the localization of a small intra-ocular foreign body, because often the size of the foreign body is smaller than the diameter of the effective focal spot and the x-ray tube. This causes a dense central shadow, or umbra (fig. 3 *A*), and a peripheral zone of decreased density, or penumbra (fig. 3 *B*). As the penumbra is not visible through the bony structures it may be readily understood that many minute particles are rendered invisible on the x-ray film, irrespective of their density.

Axiom 2. In the localization of an intra-ocular foreign body the greatest care must be taken to secure perfect immobilization of the head and eye, use of the smallest aperture practicable for the emission of the beam of the roentgen rays and the finest possible effective focal spot within the x-ray tube to be used.
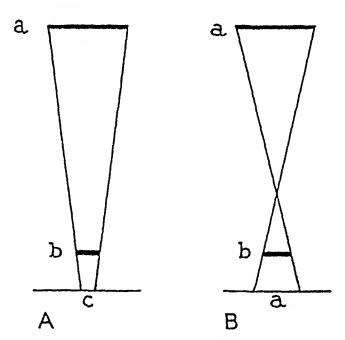
Fig. 3.—A, diagram illustrating the umbra phenomenon, in which a, indicates the focal spot of the x-ray tube; b, the foreign body, and c, the x-ray film. The foreign body is smaller than the diameter of the focal spot, or source of radiation. The resulting image on the surface of the film (c) is smaller than the foreign body (b). B, diagram illustrating the penumbra phenomenon, in which a, indicates the focal spot; b, the foreign body, and c, the size of the penumbra. The penumbra is larger than the size of the foreign body and causes a zone of relatively decreased density surrounding the actual image, or umbra.
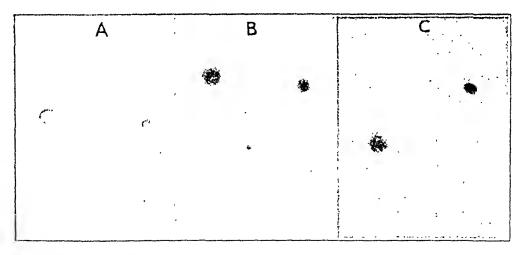
Fig. 4.—A, roentgen ray projection of three lead particles, with the x-ray tube at a distance used with the modified Sweet localizing apparatus. The focal spot of the tube is approximately 1.1 mm. in diameter. The distance of the foreign body from the surface of the film is approximately the same as in the actual localization practice on the patient. B, roentgen projection secured under conditions similar to those for A, except that a focal spot of 3.2 mm. was used. C, roentgen projection secured under conditions similar to those for A, except that a focal spot of 5.5 mm. was used. Notice the effect on the outline of the foreign body. As the focal spot increases, the outlines become indistinct.

GEOMETRIC LOCALIZATION

A discussion of the underlying principles used in the geometric methods of localization of an intra-orbital foreign body is essential to an intelligent understanding of the problems involved. I seek especially to bring out those factors which cause errors in accuracy so that the ophthalmologist may be on his guard in criticizing the results obtained by any of these methods. The most commonly understood and generally accepted geometric method is either the original method of Sweet or the more recently evolved modified Sweet procedure. The apparatus in either case is built as an instrument of precision and embodies in its use many factors with which the roentgenologist must be thoroughly familiar. This is understood in the larger clinics, where trained roentgenologists see abundant material, but in the small medical centers, where a case of an intra-orbital foreign body is met with only occasionally, it is more difficult for the radiologist to familiarize himself with the apparatus and method sufficiently to understand its strong and weak points. These I hope to bring out in the following discussion.

To define the position of any point in space with relation to another, three coordinates are required, corresponding to the three geometric planes in space. In considering the globe of the eye I refer to these three planes as the equatorial diameters and name them, according to anatomic terminology, the horizontal, the sagittal and the coronal plane. The intersection of these planes is the optic center of the globe, and a knowledge of the distance of the foreign body from this optic center in relation to the three spatial planes definitely localizes it in relation to the ocular structures. The modified Sweet method will be discussed to illustrate the principles of geometric localization, but these principles apply to all geometric methods. In this procedure of localization it is presupposed that the eyeball remains stationary during the examination, and a marking device is placed in a fixed and known relation to the optic center. In the modified Sweet apparatus this is done by accurately centering a small metallic marker directly in the line of the optic axis, which is the center point of the pupil, and exactly 10 mm. anterior to the tangential plane of the cornea. The mechanical construction of the instrument assures this constant relationship of the marker to the globe. It also embodies in its construction a constant distance between the target of the x-ray tube and the surface of the film, a constant inclination of the beam of the roentgen rays and a constant shift of the tube, or the formation of a parallax angle. With these factors known and predetermined, one is able to define the position of a foreign body in relation to the marker and calculate its relation to the optic center.

Two views are made. The first is taken with the target of the x-ray tube in exact alinement with the marker, which allows one to measure

accurately the distance of the foreign body posterior to the marker and thus determine its distance anterior or posterior to the coronal equatorial plane. The same view allows one to determine the distance above or below the horizontal equatorial plane which is also indicated by the position of the marker. Only one more measurement is required to identify the position of the foreign body within the globe, this measurement being the distance to the temporal or nasal side of the sagittal equatorial plane. To obtain this the x-ray tube is shifted parallel to the surface of the film a predetermined distance, and a second exposure is made. The parallax shift forms two series of triangles, in one of which series the marker is the apex of the triangles, the foreign body being the apex in the other series. The series in which the marker is used may be illustrated by figure 5, in which the distance of the marker from the surface of the film can be easily determined by the formula $X = \frac{BC}{A + B}$. The series in which the foreign body is used as the apex may be illustrated in a similar manner, the distance of the foreign body from the surface of the film being calculated by the same formula, as this formula applies to the height of similar triangles, irrespective of their inclination. It is therefore possible to determine accurately the position of the foreign body in relation to the temporal or nasal side of the sagittal equatorial plane. As these planes intercept at the optic center, one has the relation of the foreign body to the optic center, and the localization is complete.

It now remains to chart the findings so that there will be a permanent record. It will be noticed that the chart reconstructs the actual operating conditions and factors embodied in the apparatus for localization. Figure 6 shows the apparatus as viewed from the end and indicates the position of the marker, the inclination of the beam of the roentgen rays and the position of the film in relation to the eyeball. Measurements made from the x-ray film are indicated on the chart in column E. These measurements, however, are not in millimeters, because of the magnified distortion, but are made in arbitrary units which correspond to millimeters. The key plate used to measure these arbitrary units is a handy, convenient and accurate manner of transferring them from the film to the chart. Similar measurements are made, the columns indicated across the top of the key plate being used to transfer to corresponding columns on the localization chart. Lines are retraced parallel to those indicating the beam of the roentgen rays and where these cross the foreign body is located. Thus one has established a certain set of known and predetermined conditions in using the instrument for localization and has merely reversed these conditions in charting the results of the procedure. In all geometric methods this general type of procedure is followed, with various minor modifications.

There are several possible sources of error inherent in the apparatus, such as deviation in alinement of devices for centering, carelessness in perfect centering and alinement of the tube, slight movement of the
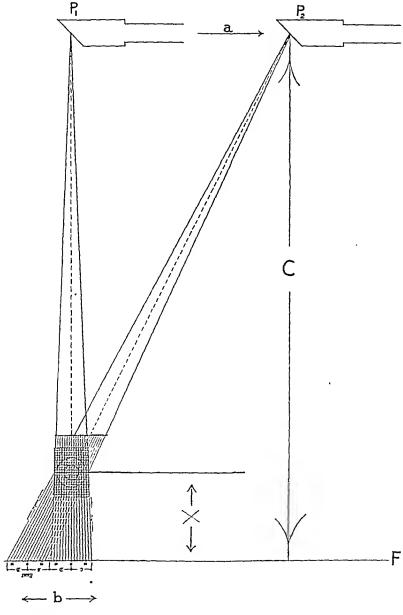
Fig. 5.—Relation of the localization chart to the x-ray tube as viewed from above. The tube is shifted from the point P_1 to the point P_2. a represents the direction of the shift; b, the parallax shift of the image on the x-ray film; C, the distance of the x-ray tube from the surface of the film (F), and X, the distance of the marker from the surface of the film.

apparatus while shifting the tube and inability of the patient to maintain ocular fixation, especially in cases of nervousness, photophobia or injury of the eye. The procedure must be carried out with meticulous

care and repeated if there is any question as to the findings. We always insist that two workers make independent measurements and charts, which must agree perfectly before the results are acceptable.

If the foreign body is small, a single central point is chosen, but if it is sufficiently large, two points, usually anterior and posterior, are localized, different colored inks being used.

In the procedure just described the foreign body has been localized, and its position has been indicated on the chart provided for this purpose (fig. 7), which represents the usual type of chart used in the
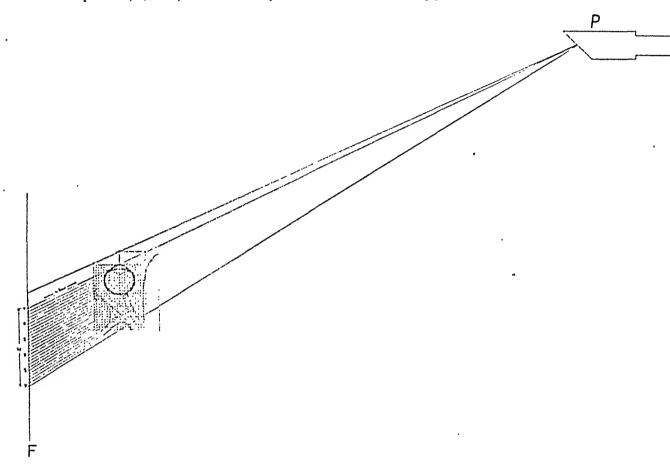
Fig. 6.—Relation of the localization chart to the x-ray tube as viewed from the side, indicating the angle of inclination of the beam of the roentgen rays from the point P and the position of the eyeball in relation to the metallic marker and to the surface of the film (F).

geometric methods of localization and specifically in the modified Sweet method. It must, however, be remembered in examining this chart that the position of the foreign body has been indicated in reference to a model eye of a diameter of 24 mm. and that this is not necessarily the position of the foreign body within the eye of the patient. The actual eye may be larger or smaller than the model eye. It is more often smaller, because of trauma or partial collapse due to loss of aqueous. Therefore, if the foreign body appears to be outside of the mottled

globe on the localization chart it is probably outside the model globe. A greater fallacy is present when one assumes the foreign body to be actually in the eye because of its apparent position within the model eye as indicated on the localization chart.

Some assistance may be obtained by examining the eye with the ophthalmoscope or, if this is impossible, owing to hemorrhage, trauma or opacity of the media, the opposite eye may be examined. In this
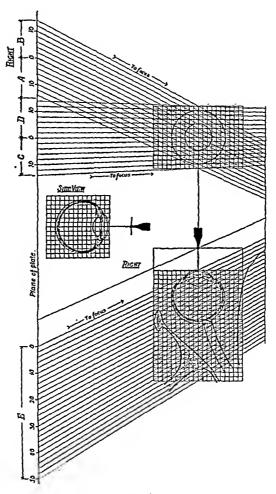
Fig. 7.—Chart used in the modified Sweet method of localization of a foreign body in the eye. These diagrams are typical of the geometric methods of localization. Entire localization is done in reference to the front and horizontal diagrams of the eyeball. The side view is not necessary but aids one to visualize the position of the foreign body.

manner one determines whether the eye is myopic or hyperopic, and this is an aid in deciding whether the actual eye is larger or smaller than the model eye.

The foreign body may, however, appear to be located within the globe on all three of the diagrams of the localization chart and yet

may be actually beyond the limits of the model globe.[6] This is possible because only the equatorial diameters are shown on the chart. As a means of clarifying this problem let it be supposed that, as illustrated in figure 8, an x-ray tube is pointed toward the pupil, the central beam being indicated by the arrow *b*. The image of the globe on the x-ray film *A* would appear circular, and the diameter of this circle would be equal to the greatest diameter of the globe. In this theoretical instance spreading of the roentgen rays is not considered, but they are thought
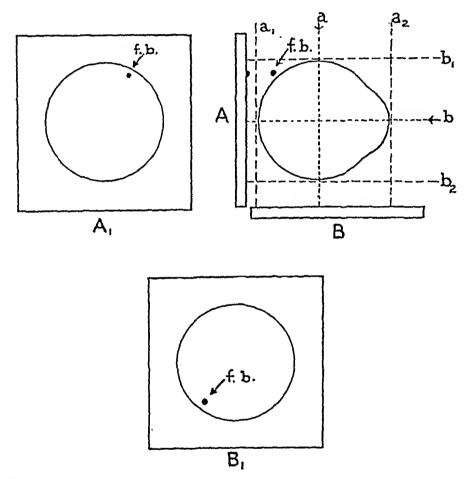
Fig. 8.—If a foreign body (*f.b.*) is beyond the limits of the globe, it may appear to be inside the globe in the anterior projection (*A*) if the beam of the roentgen rays *b* is directed as shown. A_1 would be the resulting image on the x-ray film. It would also appear on a film made at a right angle to the former (*B*), because the beam of the roentgen rays *a* projects the image of the globe as a circle equal to its greatest diameter, and B_1 would be the resulting image on the x-ray film.

of as a parallel beam of rays. If, therefore, a foreign body is in close relation to the posterior border of the sclera but not actually within the globe, it would be projected on the x-ray film A_1 as though

6. Stephenson, F. B.: Sweet Improved Eye Localizer Chart: How to Avoid Possible Misinterpretation, Am. J. Roentgenol. **16**:470 (Oct.) 1926

it were within the globe. By making a similar projection at an angle of 90 degrees it is possible to see that the foreign body could appear to be within the globe on both x-ray films and yet actually be outside, because the image of the eyeball has been on the x-ray film as the circle which shows only the greatest, or equatorial, diameter. In a similar manner it is easy to conceive that a third projection made in the other spatial plane might easily show a similar phenomenon. This is an actual error in many cases on the geometric localization chart. It may be argued that one does not make the projections for localization in this manner, but it must be fully understood and always remembered that the final result as reconstructed on the chart is visualized as though the projections of the roentgen rays were actually made in the three spatial planes, and the model globe is indicated as though it were actually visible. One must therefore be especially cautious in interpreting the appearance of a foreign body which seems to be in close relation to the posterior border of the sclera as within the globe, even though it may appear to be within the globe in all three diagrams on the chart. If the foreign body lies in close relation to one of the equatorial planes as indicated on the diagrams, there is less likelihood that the fallacy will enter, but when the foreign body appears to lie nearly midway between the equatorial planes one is likely to be led astray in assuming it to be within the globe. If one imagines the circles indicating the globe to be bounded by squares, the positions in which the greatest error is likely to occur are near the corners of these squares.

It is therefore of the utmost importance in this type of work to determine exactly the position of the foreign body in relation to the boundary of the sclera, since there is a greater likelihood that it lies definitely outside of the actual eye in those cases in which one can show it to be outside of the model eye. The manner of determining this important point will be presented next.

ACCESSORY METHODS

It has been shown that in using a geometric method of localization one is dealing with a model eye and localizing the foreign body in relation to the structures of this model. However, on the localization chart one views this model as though it were projected in the three equatorial diameters, and it is well to remember that the foreign body may appear within the model globe in all the diagrams on the chart and yet be actually outside this globe.

To ascertain if such is the case it is well to examine the position of the foreign body in relation to the diagram which illustrates the side view, or sagittal projection, of the globe. It is to be imagined that the diagram of the globe is enclosed in a square, the lines bisecting the

square overlying the horizontal and coronal equatorial planes. A foreign body lying in close relation to either the horizontal or the coronal equator is less likely to be beyond the limits of the model globe, but a foreign body which lies near the scleral boundary almost an equal distance between these equators is probably outside the limits of the globe. We are not so intimately concerned with those foreign bodies which lie in the regions of the anterior corners of the square, as the localization under discussion is considerably more accurate for a foreign body in the anterior hemisphere. Furthermore, if there is any doubt as to whether a foreign body which appears to lie in either of these positions is intra-ocular, a thorough search of the eyelids and the conjunctiva and soft tissue of the globe will in many cases settle the question. In the posterior positions, however, the question of double perforation arises and offers considerably more difficulty.

It remains to determine the actual plane in which the foreign body lies and to transfer this measurement to the appropriate diagram. This is done on the front view, or the coronal section. The procedure consists in bisecting the foreign body with a line extending to the scleral boundary. It may be done in either of two planes, horizontal or sagittal. If it is done in the horizontal plane, the measurement must be transferred to the horizontal projection; if it is done in the sagittal plane, the measurement must be transferred to the side view.

The measurement of the actual plane of the foreign body is then used as the diameter of a circle the center of which is the optic center of the globe. If the foreign body lies beyond the limits of this circle in either projection, it is outside the model globe. It is, of course, possible to transfer a similar measurement from any one projection to either of the others, but we use the front view as a routine to measure the planes as a matter of simplicity.

Figure 9 illustrates the method. Diameter *A* measures the actual width of the globe on a level with the foreign body in the horizontal plane. This is measured by calipers and transferred to the horizontal section as the diameter of a circle. The foreign body lies definitely beyond the boundary of the circle and is therefore extra-ocular.

Diameter *B* measures the actual width of the globe on a level with the foreign body in the sagittal plane. This is transferred in a similar manner to the side view, but the foreign body lies so close to the circle that one cannot be certain that it is extra-ocular. It must be remembered, however, that it is necessary for the foreign body to be outside the resulting circle in only one projection to be definitely beyond the limits of the model eye. In a similar manner, the diagnosis of an extra-ocular foreign body is made if the particle appears beyond the limits of the sclera on any one of the diagrams. The procedure just described appears to be simple and accurate, yet it is surprising

how many times in actual practice it will not definitely answer the question. In such cases one has a further check by mathematical means (fig. 10). The foreign body is known to lie a certain distance from each equatorial plane. *A* is the distance anterior or posterior to the coronal equatorial plane; *B,* the distance to the temporal or nasal side of the sagittal equatorial plane, and *C,* the distance above or
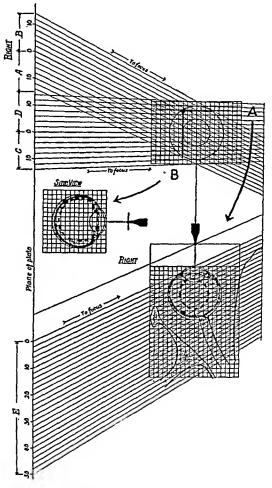
Fig. 9.—Method of transferring the actual diameter on which the foreign body lies to the appropriate diagram. Diameter *A* is parallel to the horizontal equatorial plane and must therefore be transferred to the horizontal view in the diagram. The result shows the foreign body to be definitely beyond the limits of the globe. Diameter *B* is parallel to the sagittal equatorial plane and must therefore be transferred to the side view. Judging by this result, there is no doubt that the foreign body is extra-ocular. Special care is not required in drawing these circles, as the entire procedure is intended only as an approximation.

below the horizontal equatorial plane. One then constructs in imagination a rectangular figure, the diagonal (*X*) of which would be the actual distance of the foreign body from the optic center. This is calculated by taking the square root of the sum of the three given

diameters according to the formula $X = \sqrt{A^2 + B^2 + C^2}$. The radius of the model eye is known to be 12 mm. Therefore, if the resulting figure for X is over 12, the particle is extra-ocular. In practice this is simplified by squaring the three diameters and adding them. If this gives more than the square of 12, or 144, the foreign body is extra-ocular; if it gives less, the foreign body must be intra-ocular.

The exact relation of the foreign body to the model eye has been determined, but this does not place the foreign body in anatomic rela-
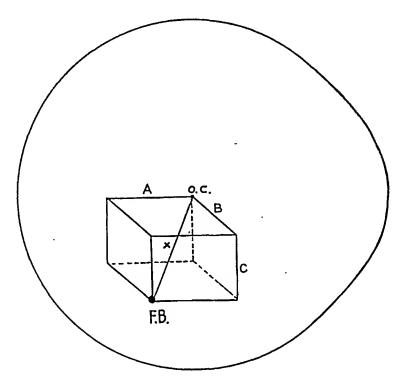
Fig. 10.—Diagram illustrating the method of determining by mathematical means whether a foreign body is intra-ocular or extra-ocular according to the formula $X = \sqrt{A^2 + B^2 + C^2}$. *O.C.* indicates the optic center; *F.B.*, the foreign body; *A*, the measurement posterior to the coronal plane; *B*, the measurement to the temporal or nasal side of the sagittal plane; *C*, the measurement below the horizontal plane, and *X*, the diagonal of the rectangular parallelopiped.

tion to the eye of the patient. To accomplish this two procedures are used—bone-free roentgenography of the anterior hemisphere and the parallax method.

If the foreign body appears to be in the anterior hemisphere and cannot be seen on ophthalmoscopic or slit lamp examination, it may often be shown by a bone-free projection.[7] This is made by bending a dental film and slipping it between the eyelids and the bony orbital

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7. Butler, T. H.: Bone-Free Radiographs, Proc. Roy. Soc. Med. (Sect. Ophth.) **17**:6, 1923-1924. Lindblom, K.: Bone-Free Radiography of the Eye, Acta radiol. **15**:615, 1934.

margin (fig. 11 *A*). The beam of the roentgen rays is then directed at an angle of 90 degrees or slightly greater toward the film, the central ray being aimed at the outer canthus. A very soft radiation is used, and the film (fig. 11 *B*) shows the cornea and anterior portion of the globe between the lids. In this manner assistance .is given in ascertaining the position of the particle and in recognizing some particles which are so small or have a specific gravity so low that they cannot be seen on the usual projections. It is essential to have the lids opened as widely as possible to show the globe to advantage. We have in a few cases recognized a foreign body buried in the eyelid by this means. Some workers trim down the x-ray film and slip it
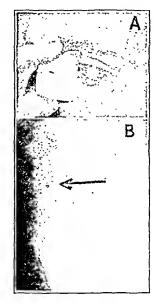
Fig. 11.—*A*, method of holding a dental film for soft tissue projection of the anterior portion of the globe. It is essential to have the eyelids separated as widely as possible. In practice an assistant holds the eyelids apart. *B*, roentgenogram taken by the method of soft tissue projection, showing the foreign body.

into the conjunctival sac; others make similar projections from above, but we have not found that these practices add anything essential and believe that projection with the use of a dental film is sufficient for all routine work.

The parallax method [8] is based on the principle that if a foreign body is intra-ocular it will rotate about the optic center when the eye is directed between predetermined external points of visual fixation. Thus the rotation of the eye becomes the factor of importance in determining whether a particle apparently within the model eye is also in the actual eye or is outside.

8. Verwey, A.: Localization of Foreign Bodies in the Eye in Relation to the Rotation Center, Am. J. Ophth. **7**:337, 1924.

The patient is placed with the side of the head against the x-ray film and directed to gaze at a spot on a level with the eye. A film is exposed, and another film is placed in position without the position of the head being disturbed. This requires some sort of film-holding device. The patient is instructed to look at a point above the first position by rotating his eyes upward but is cautioned not to change the position of his head. Another film is exposed, and a third is placed in position. The patient then rotates the eye downward to a point an equal distance below the horizontal meridian, and a third film is exposed. In routine practice we make three heavy marks on a yard-stick, the upper and lower points being equal distances from the
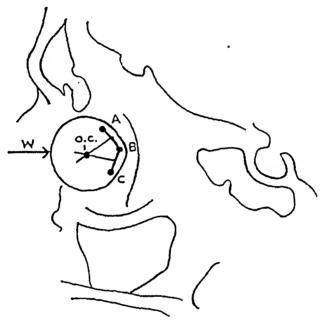
Fig. 12.—Tracing made by superimposing the x-ray films. *O.C.* indicates the optic center; *A, B* and *C,* images of the foreign body as the eyeball rotates between the points of visual fixation, and *W,* wire indicating the optic axis and the tangential plane of the cornea.

midpoint. This is supported about 4 feet (1.2 meters) in front of the patient, with the middle marker on a level with the eyes.

One now has three films which, after being processed and dried, may be superimposed. There will be three images of the foreign body present, if it has moved owing to the rotation of the globe. One makes a tracing of the films by superimposing a sheet of thin paper and drawing the main anatomic marks and the image or images of the foreign particle and adds to this the approximate position of the globe. This is done by placing across the bridge of the nose, before the films are exposed, a straight fine wire, held on by adhesive tape in such a manner that the end of the wire is adjacent to the tangential plane of the cornea and centered at the midpoint of the pupil as judged from the lateral aspect (fig. 12). This wire is shown on all three roent-

genograms and not only insures accuracy in superimposing but indi-
cates the tangential plane of the cornea and the level of the optic axis.
The eyeball is now roughly sketched in, and one has a drawing similar
to figure 12.   The optic center can be indicated with reasonable cer-
tainty, and it may be determined whether the particle rotates about
this center by connecting the three images of the foreign body with
straight lines and dropping ordinates from these lines.   If the ordinates
cross at or near the optic center, the foreign body is intra-ocular; if
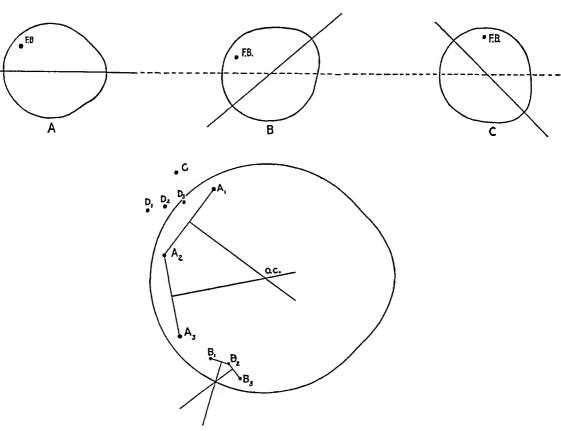
Fig. 13.—*A,* position of a foreign body (*F.B.*) in the globe with the eyeball
directed to the horizontal point; *B,* position with the eyeball rotated upward; *C,*
position with the eyeball rotated downward. A_1, A_2 and A_3 show the behavior of
an intra-ocular foreign body; B_1, B_2 and B_3, the movement of an extra-ocular
foreign body (*C*), and D_1,D_2 and D_3, the behavior of a foreign body which has
become embedded in the superior rectus muscle.

they cross at any other point either within or outside of the globe, the
particle is extra-ocular.   This is illustrated in figure 13, which shows
the behavior of both types of bodies.

In the great majority of cases of an extra-ocular particle the move-
ment is nil or so slight that there is no question as to the position of
the particle.   Two sources of error may be present.   If a body that
is extra-ocular is embedded in Tenon's capsule or is adherent to the

posterior part of the sclera it may behave as though it were within the eye, and a foreign body in an unusually fluid vitreous may behave as though it were extra-ocular. Thus it may be seen that all problems of localization of an intra-ocular foreign body cannot be accurately settled by this means, but it is of great advantage in those cases in which the particle is in close relation to the posterior part of the sclera. I have previously stated that localization by geometric methods is less accurate in the posterior portion of the globe, and the ability to localize a foreign body here is the greatest advantage of the so-called parallax method as a check-up procedure.

Another possible problem may arise in cases in which it is necessary to decide whether the foreign body is intra-ocular when it lies near the lateral poles of the eye and does not show sufficient rotation about the axis for localization by the method described, owing to its close proximity. We have in cases of this condition made postero-anterior projections in a similar manner to that described, the eyes being rotated laterally.

Thus many problems arise in the routine localization of intra-orbital foreign bodies which require various methods supplementary to the standard geometric procedures. It is possible to determine with considerable accuracy the exact position of the particle in relation to the structures of the patient's eye. In a few cases there still remains a doubt, and further check-up studies are required. These problems will be discussed in the final section.

VISIBILITY OF FOREIGN BODIES

It is important for the ophthalmologist to familiarize himself with the methods of the roentgenologist, but it is equally advantageous for the roentgenologist to be acquainted with the problems as they present themselves to the operating ophthalmologist. If a foreign body is responsive to the magnet, the operator will usually try to extract it through the wound made by its entrance. If the foreign body is in the vitreous, it is of great importance in many instances to make a check-up localization to determine how far it has been displaced, if the operator has not succeeded in removing it. If it cannot be drawn through the ciliary body or if it adheres to the posterior capsule of an undamaged lens, it is often better to do a sclerotomy rather than continue to use magnetic traction in the original direction. If it is embedded in, or becomes firmly fixed to, the iris, it is often better to do an iridectomy. If the foreign body is large and is in the vitreous, especially in a case in which the lens is clear or the condition has been of long standing, with organization of material about the particle, it may often be extracted through a posterior sclerotomy. If

it is in the lens, noninterference is sometimes the best policy until cataract develops. If the particle seems to be buried in the sclera and the eye is quiet, many operators do not disturb it. Others give a single trial with the magnet and send back the patient for relocalization of the foreign body. If there is no movement, they go no further. If there is a double perforation and the eye is quiet, the foreign body is usually not disturbed. If the body is nonmagnetic, it may sometimes be removed by searching blindly and sometimes by biplane fluoroscopic control. If it is not removed, it is a potential source of danger not only to the injured eye but also to the good eye, and if there is any doubt, enucleation should be seriously considered.
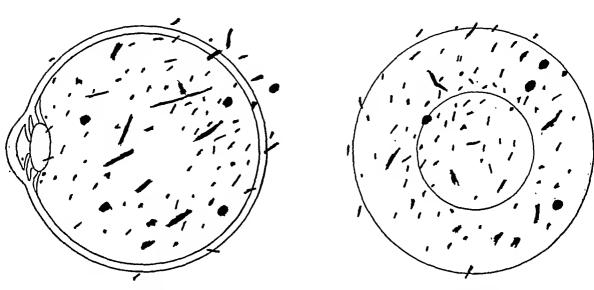
Fig. 14.—Relative size and position of the intra-orbital foreign bodies in one hundred unselected cases, as shown by geometric localization. There might be a question of double perforation in from twenty to twenty-five of these cases.

We recommend that all roentgenologists be familiar with the simple problems just mentioned and that they follow the patient when possible or at least learn the manner and results of the operative procedure. This will greatly aid them in acquiring a clearer understanding of the problems involved. I have shown diagrammatically (fig. 14) the relative size and position of the particles in one hundred unselected cases observed in the roentgen department at the Wills Hospital without reference to which eye was involved. The drawings illustrate the position as shown on the localization charts. It will be noted that there arises a possible question of double perforation in about twenty to twenty-five of the cases. It appears that in one case the foreign body was buried in the posterior part of the sclera. Actually many of these eyes were so badly infected that enucleation was the procedure of choice. I would estimate that in at least 10 per cent of the routine cases this

question of double perforation has arisen. In nine of the ten cases the question can be settled by the previously described procedures, but there still remain a few cases in which the condition is puzzling in spite of all attempts to obtain complete evidence. In these cases we resort to the injection of air into the capsule of Tenon and auto-visualization.

The injection of air into Tenon's capsule is made in an attempt to render the posterior hemisphere visible on roentgen examination. Some workers have used opaque material, but we have had such good results with air that we believe it to be preferable. Iodized poppy-seed oil 40 per cent, diodrast and other opaque mediums remain for a long period after injection and tend to prevent clear visualization of the foreign particle. Air appears to diffuse or absorb quickly, has been perfectly safe and harmless thus far and aids rather than prevents visualization of the particle. We do not think that sufficient evidence has been gathered as yet to recommend the practice promiscuously, and it should never be attempted by the radiologist alone. However, when the question of double perforation arises and cannot be settled by other means, especially if judgment as to an operation hinges on the result, we have not hesitated to use the following method.[9]

The eye is thoroughly anesthetized. The patient is then instructed to gaze downward and inward, turning the injured eye toward the tip of the nose. A point about midway between the superior oblique muscle and the external rectus muscle is chosen, and the eye is fixed in position. A needle is bent at an angle of about 45 degrees and inserted below the conjunctiva about 4 mm. anterior to where the reflection of Tenon's capsule is thought to be. It is now introduced posteriorly from 3 to 4 mm. under the conjunctiva until it is sufficiently far back to be over the capsule (fig. 15). By rotation the point is turned downward, and the capsule is punctured. It is essential to bend the needle and proceed by the technic just described, because the air injected would run directly back about the barrel of a straight needle and escape. A syringe containing from 8 to 10 cc. of air is then attached, and the injection is made gently but steadily. When about 6 cc. has entered, there are an increase in resistance and a slight but distinct proptosis. The proptosis is a guide to the correctness of the procedure. If the needle is not in Tenon's space the injected air will cause a bulge in the conjunctiva without proptosis. It is best in this case to discontinue the injection for about forty-eight hours and then try the procedure again. The patient should complain of little or no discomfort.

9. Spackman, E. W.: X-Ray Diagnosis of Double Perforation of the Eyeball After Injection of Air into the Space of Tenon, Am. J. Ophth. **15**:1007 (Nov.) 1932.

X-ray films are exposed at leisure, and the processing is done immediately to be sure that the results are satisfactory. It is a wise precaution to make a single lateral projection before the injection to compare with subsequent projections. The air in Tenon's space may form a contrast layer (fig. 16 *A*) or a sharply demarcated band (fig. 16 *B*), which should be readily distinguished on the film. It is advisable to make exposures both with and without the use of the Potter-Bucky diaphragm, but casettes and intensifying screens should always be used. A single lateral projection is not to be depended on, unless the foreign particle is definitely seen to be extra-ocular. The same error can be easily made as described in discussing the charts used in the geometric methods of localization, and the foreign body appears to be within the globe because of superimposition. There must be a sufficient number of exposures made from various angles so that the
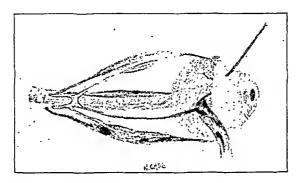
Fig. 15.—Drawing illustrating the method of injecting air into Tenon's capsule. The bent needle is inserted from 3 to 4 mm. under the conjunctiva before penetrating Tenon's capsule. By going through two layers in this manner there is less likelihood that the injected air will escape.

beam of the roentgen rays meets the posterior sclera tangentially at the point where the foreign particle lies. This is done by gradually rotating the head and making serial exposures after each slight movement. The direction of rotation can be determined by examining the localization chart and by bearing in mind the fact that the image of the particle appears to approach the scleral edge after rotation if the rotation is in the right direction.

By this method a final diagnosis of double perforation may be verified or disproved. This procedure is not successful in all cases, however. Occasionally an extremely nervous patient will cause difficulty, and in the case of other patients we found that injection was not possible. We believe that in cases in which the latter was true the difficulty was due to adhesions or fusion between the capsular layers.

The patient is comfortable afterward, and the air is completely absorbed in from two to four days.

One other method of check-up is used and is being developed at present, but too few data are as yet available to enable one to be certain of its value. This we call autolocalization. Pirie[10] found that after complete relaxation of accommodation it was possible to visualize directly the roentgen rays without aid. Contrary to general belief, the retina is somewhat sensitive to the wavelength of the roentgen rays. I therefore place the patient before the x-ray tube in a darkened room and ask him whether he can see a black spot within the greenish fluorescent light which is visible when the tube is energized. If so,
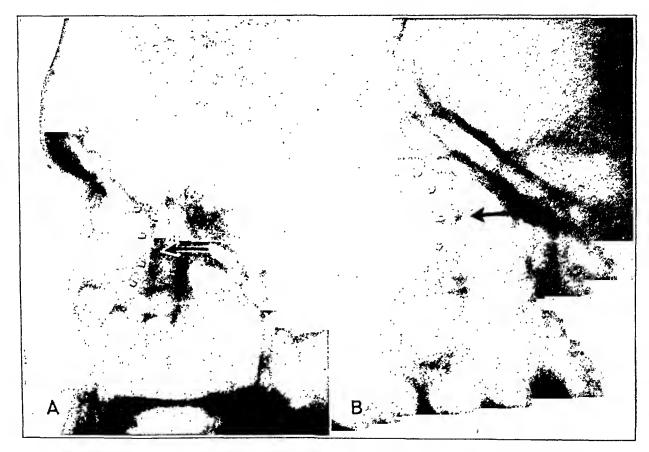
Fig. 16.—Sometimes the air causes a zone of contrast (*A*), and at other times it causes a band of decreased density (*B*). In both the cases illustrated the foreign body is shown to be extra-ocular.

he is asked to describe it and to draw its relation to cross-wires which are placed in a frame before the injured eye. A very narrow beam is used so that no more exposure than is absolutely necessary is given. The patient is then asked to draw a diagram of what he saw, placing the spot in relation to the cross-wires. He will always place it in the quadrant diametrically opposite that where it actually is, since there is no reversing action of the lens to the roentgen rays but it is

10. Pirie, A. H.: An Apparatus for Reading with Closed Eyes, Brit. J. Radiol. **7**:111 (Feb.) 1934.

interpreted as though it were reversed, as is usual with the beam of light. I have apparently shown thus far that the foreign body, if small, must be within a few millimeters of the retina to be seen and that it can be placed by the patient with considerable accuracy, which helps to check the data for localization. In cases in which the foreign body has perforated the retina, we have failed to obtain results with this procedure, and therefore we believe that this helped us in several instances in which the foreign body had perforated or was buried in the sclera. Between thirty and forty patients have been tested in this manner, and this method is mentioned for the benefit of those workers who have the opportunity to try it. Complete relaxation of accom-
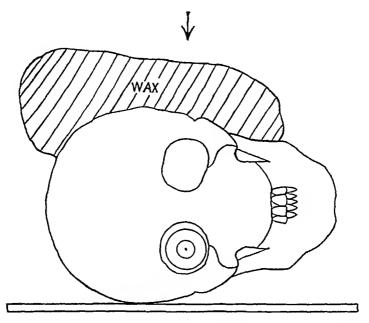
Fig. 17.—Method of approximating the density of the human head as noted roentgenographically by using a dried skull and wax. An enucleated eye was placed within the skull, and wax was added until experimentally a roentgenographic effect was obtained similar to that obtained with the average human head.

modation is essential; the patient must remain in total darkness between fifteen and twenty minutes before the attempt is made. Warning must also be given to avoid overexposure, as it is easy to forget this precaution.

We have sought to show experimentally the relation between the size, the specific gravity and the density of the tissue as applied to the problem of the visualization of intra-orbital foreign bodies. It has long been recognized that the roentgen visibility of a body is directly proportional to the specific gravity. We therefore collected samples of all materials which have been found to act as intra-ocular foreign particles in the cases observed by us during the past twelve years and proceeded empirically to build a model of actual working conditions (fig. 17). A dried skull was coated on one side with paraffin until
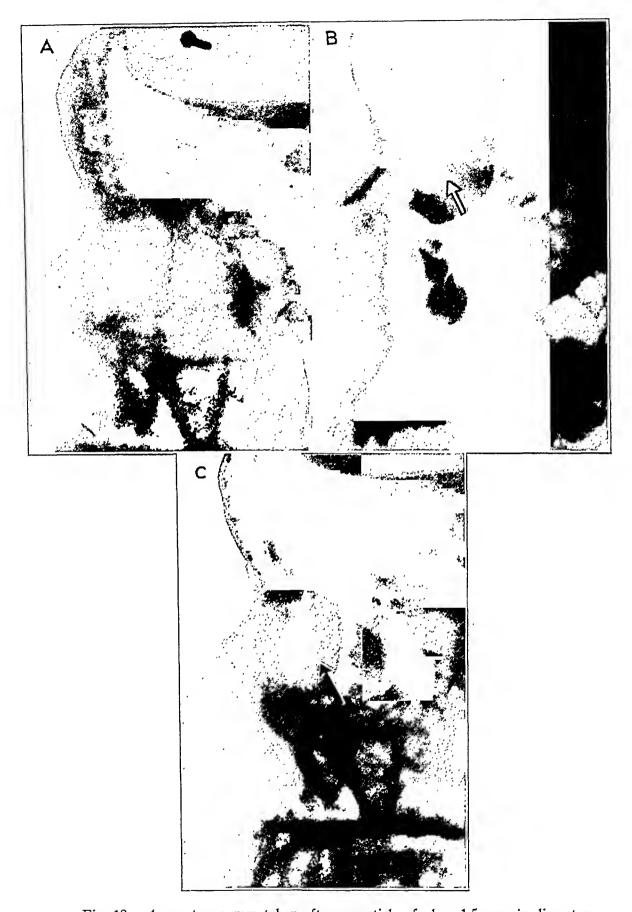
Fig. 18.—*A*, roentgenogram taken after a particle of glass 1.5 mm. in diameter, with a specific gravity of 3, was embedded in an eye which was placed in the skull. This is not visible on the x-ray film. *B*, roentgenogram taken after a particle of aluminum 2 mm. in diameter, with a specific gravity of 2.5, was embedded in a similar manner. This is barely visible through one layer of bone. *C*, roentgenogram taken after a particle of marble 3 mm. in diameter, with a specific gravity of 2.8, was embedded. This is visible through one layer of bone.

lateral exposure through it gave a roentgenogram showing the same average quality, detail and density that are usually obtained with the head of a living person. The paraffin afforded approximately the same degree of absorption and scattering as human tissue. An enucleated eye was then placed in the orbit, and we proceeded to embed within the eye particles of the various substances. These were chosen by crushing the material and obtaining roughly spherical bits which ranged in size on measurement with a magnifying glass and calipers from 0.5 mm. upward in steps of 0.5 mm. These were sorted, arranged and tested in the manner already described, the larger particles being tested first, until the limit of visibility through the orbital bones was reached (fig. 18). This limit was noted and checked, and further studies were carried out by embedding the particles within the eyeball after its
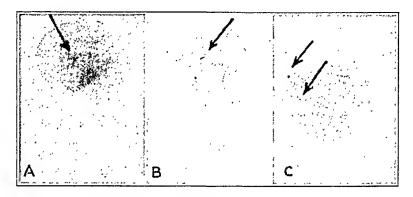
Fig. 19.—Results obtained by placing the enucleated eye on the x-ray film and embedding a foreign body within it. These are similar to those obtained with bone-free roentgenograms. *A,* roentgenogram showing a particle of lead less than 0.5 mm. in diameter, with a specific gravity of 11.3. This was deeply embedded and exposed to fairly dense radiation. *B,* roentgenogram showing a particle of steel less than 0.5 mm. in diameter, with a specific gravity of 8.5. This was deeply embedded, and radiation of lesser density was used. *C,* roentgenogram showing two particles of emery less than 0.5 mm. in diameter, with a specific gravity of 4, one of which was placed on the surface of the eye and the other embedded. The embedded particle is barely visible on the film.

removal from the skull. In this manner we obtained data which gave us the approximate size a foreign body of a given specific gravity must be to be visualized by the usual method of localization and also by the bone-free projection (fig. 19). We noted the results as: (*a*) visible through bone, (*b*) questionably visible through bone, (*c*) visible through soft tissue and (*d*) questionably visible through soft tissue. It is realized that this is only a relatively rough guide, as superimposition of bony layers in the orbit would obscure some particle which would be plainly visible in other positions. However, it acts as a

fair guide and has been found to be accurate during the past few years. By referring to the chart the ophthalmologist may obtain some idea of the required size of an intra-orbital foreign body before it can be shown on roentgen examination. He may often estimate to a certain extent the probable size of the foreign body by the patient's statement, the size of the wound caused by the entrance of the foreign body and other evidence and obtain more accurate assistance from the report
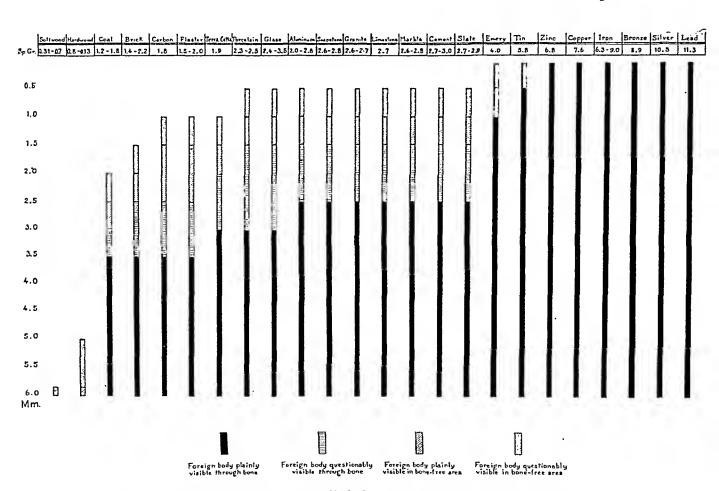
Fig. 20.—Composite chart compiled from data obtained in the experimental work described in the text, showing the degree of visibility of various particles in relation to their size and specific gravity.

of the radiologist. This is of special service in the case of particles of nonmagnetic substances. The chart (fig. 20) is a handy means of referring to the aforementioned data, and we have found it of value in many problems. In making the experiments we sought to duplicate exactly the conditions under which the work on localization is done relative to the distance of the tube from the film, the focal spot, the exposure, etc., so that it would be of assistance in checking the routine work.

SUMMARY

In regard to the localization of an intra-orbital foreign body, the roentgenologist should always familiarize himself with the history, examine the wound made by the entrance of the foreign body and obtain any other pertinent data bearing on the case. Roentgenograms for identification are sometimes taken if there is a question of lodgment in some structure outside the orbit. Special projections of the orbital apexes and the optic canal are often useful. The usual study for localization is then made, invariably supplemented by soft tissue projections if the foreign body is thought to be minute or of low specific gravity and cannot be located on the usual films. We cannot too strongly stress the importance of perfect immobilization, as very slight movements of the globe may completely invalidate the results. If the results of localization are not perfectly satisfactory, a check-up study is made.

The chart used to record the results of the geometric method of localization is examined, and if any doubt exists, geometric and mathematical measurements are made to ascertain whether the foreign body is within the model eye. The parallax study is resorted to in nearly all cases in which the particle is in the vicinity of the posterior part of the sclera. If doubt still remains and it is important to determine whether double perforation is present, roentgenography after the injection of air into Tenon's capsule and autovisualization are attempted. Reliance is not placed on any one study, but all must be considered together. The report to the operating ophthalmologist includes a description and the results of all the studies made and the judgment of the roentgenologist in interpreting the data obtained.

ISOLATION OF VERHOEFF'S LEPTOTHRIX IN A CASE OF PARINAUD'S SYNDROME

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The type of the so-called clinical syndrome of Parinaud which my associates and I have seen most frequently is undoubtedly that which Verhoeff showed to be due to an infection with leptothrix and which might reasonably be excluded from syndromal grouping and referred to as a separate entity by a name of its own, such as "leptothrix conjunctivitis of Verhoeff." Although the literature gives tuberculosis of the conjunctiva a place in this group, it ought not to be difficult clinically to differentiate between the two conditions. In leptothrix conjunctivitis of Verhoeff there are an acute febrile onset associated with the conjunctival inflammation and a rapid enlargement of the preauricular, parotid and submaxillary glands. The palpebral conjunctiva shows marked swelling—a solid swelling due to cellular infiltration—and is intensely red, but the bulbar membrane shows a relatively scanty discharge or chemosis. The surface varies from a finely granular one with regular folds due to the swelling to a nodular or papilliform surface. The characteristic small semiopaque areas seen here and there look as if cooked with the point of a diathermy needle. The signs and symptoms persist for a fortnight or so and then slowly subside, till normality is restored in five or six weeks. The fever shows a rise in the evening and may persist for ten days or more. The blood picture may be normal or may show leukocytosis. The cutaneous tuberculin test and the Wassermann test may be positive or negative and are therefore of little help. Although the clinical course of this disease is fairly straightforward, in certain cases it presents difficulties of clinical diagnosis to those who have not had experience in its vagaries and even to those who have. My associates and I were pleased on this account to be able to apply a relatively simple diagnostic method in a case recently observed, namely, the cultivation of Verhoeff's leptothrix from the fluid of the preauricular gland. It is by no means simple to obtain histopathologic preparations which show the organisms in the gray areas of the conjunctiva, and unless one's laboratory technic is good, failure is likely. Dr. Verhoeff sent me some of his histopathologic specimens some years ago, and with his technic positive results were obtained in the typical clinical condition. This method is, however, too

difficult for use in the laboratory of the ordinary ophthalmic hospital; the cultural method is simpler. Neither method is certain, but either is as satisfactory as, say, inoculation of animals with a portion of the affected tissue in tests for tuberculosis or a Wassermann test for

Photomicrograph of slide preparation from a pure culture of Verhoeff's leptothrix taken by Lieutenant-Colonel H. E. Shortt.

syphilis. The material was obtained from the preauricular gland in the usual way for obtaining glandular fluid under strict bacteriologic precautions as to sterility, with a needle mounted on a syringe containing a little broth (0.5 cc.). The point of the needle was moved about in the gland to a slight extent, while positive and

negative pressure were being gently made with the syringe. The cultures were made immediately by Dr. B. P. B. Naidu, of the Medical Research Department of the Government of India (Indian Research Fund Association), attached to the King Institute of Preventive Medicine, Guindy, Madras, who at the time was carrying out research work in this hospital in collaboration with me (on trachoma and in other fields). The following details were supplied by him:

METHOD

The patient was G. a girl aged 16. Material for culture was aspirated from the center of the preauricular gland by a syringe, under sterile conditions. The material was inoculated onto the surface of (1) Soparkar's heated human blood agar, (2) hydrocele agar and (3) ordinary agar enriched by a surface smear with fresh human blood. The tubes were incubated at 30 C. anaerobically. After forty-eight hours of incubation the culture tubes showed discrete colonies, a few being present on each of the three culture mediums used.

Growth was obtained in subcultures under both aerobic and anaerobic conditions; the growth was more luxuriant under anaerobic cultivation.

Smears stained by Gram's method showed a gram-positive leptothrix. Subcultures were made on Soparkar's heated human blood agar (on this medium the growth tends to colony formation). On Dorset's egg medium the growth is profuse, producing pitting and lysis; on agar the growth is orange, moist and shining. In peptone water the growth produces general turbidity, with a heavy deposit; similar characteristics are observed in nutrient broth; in dextrose broth the growth is scanty. The appearance of the colony on agar is circular and raised, with an irregular wavy fringe which is more transparent than the colony. The growth produces acidity in dextrose, saccharose, salicin and maltose and no change in lactose, dulcitol and mannitol. Other sugars were not tried. In milk it produces alkalinity.

MEDIUMS

Soparkar's medium (for the growth of influenza bacilli) was described in the *Indian Journal of Medical Research.*[1] For the convenience of those interested in bacteriology the mediums used are detailed.

Ordinary Nutrient Agar Mixed with a Preparation of Human Blood.—A medium containing 3 per cent nutrient agar is prepared according to the following formula:

Meat extract	1 liter
Peptone	20 Gm.
Sodium chloride	10 Gm.
Agar	30 Gm.

The reaction is rendered slightly alkaline to litmus by the addition of sodium carbonate. The meat extract is prepared by boiling 1 Kg. of meat with 2 liters of water for forty minutes.

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1. Indian J. M. Research **6**:418, 1918-1919.

Soparkar's Medium.—The preparation of the human blood which is added to the agar is made as follows: One volume of freshly drawn human blood is added to a flask containing four volumes of sterile citrated saline solution (0.5 sodium citrate and 0.85 per cent sodium chloride). The blood and saline solution are thoroughly mixed by shaking, and the fluid is then heated in a water bath at 66 C. for thirty minutes. The heating results in the formation of a fine brown precipitate, which is removed by filtration through sterilized filter paper. All precautions should be taken to prevent contamination. The filtrate is a clear, deep red fluid, and 1 cc. of it is added to 10 cc. of melted agar at 50 C. After mixing, the tubes are sloped and allowed to set. The medium thus prepared is clear and transparent.

Hydrocele Agar.—Fifty cubic centimeters of sterile hydrocele fluid is added to 10 cc. of melted 4 per cent nutrient agar at 48 C. The ingredients are gently mixed, and the tubes are sloped; vigorous shaking should be avoided.

Other Mediums.—The ascitic fluid agar of Morax, which gives excellent growths for many of the conjunctival pathogens in Paris, was not so useful in India, since the ascitic fluid locally available had not an optimum albumin content (from 1.5 to 1.6 per cent of the albumin content of the blood serum), nor could the "ox coagulated serum," which Morax considers an excellent medium, be conveniently obtained.

Comment.—Dr. Naidu found that with the three mediums, the formulas of which have just been given, good all-round results were obtained with the conjunctival flora, on both aerobic and anaerobic cultivation.

RELATIONSHIPS BETWEEN ANISEIKONIA AND AMETROPIA

FROM A STATISTICAL STUDY OF CLINICAL CASES

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AND

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HANOVER, N. H.

INTRODUCTION

The importance of aniseikonia as an etiologic factor in certain visual disorders has been proved by previous investigations,[1] but there are still many misconceptions[2] concerning the relationships between aniseikonia and ametropia. The object of this paper is to clarify these misconceptions by presenting a statistical analysis of the cases of 283 patients who have received definite relief from ocular discomfort by the correction of aniseikonia.

During the early years of the development of the research on aniseikonia the patients who were examined at The Eye Clinic of the Dartmouth Medical School constituted what might be called a highly selected group.[1a] At least 90 per cent of them were referred to The Eye Clinic because all other procedures used had failed to give relief or reveal any organic cause for their discomfort. In general, the symptoms

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1. (a) Carleton, E. H., and Madigan, L. F.: Size and Shape of Ocular Images: II. Clinical Significance, Arch. Ophth. **7**:720-738 (May) 1932. (b) Madigan, L. F., and Carleton, E. H.: A Clinical Report of the Correction of Differences in the Size and Shape of Ocular Images, Ann. Distinguished Serv. Fund. Optom. **1**:71-97, 1932. (c) Hughes, Wendell L.: Aniseikonia: Some Clinical Observations, Am. J..Ophth. **18**:607-615 (July) 1935; (d) Aniseikonia, ibid. **19**:686-688 (Aug.) 1936. (e) Doane, H. C.: The Clinical Significance of Differences in the Relative Size and Shape of Ocular Images, Tr. Am. Acad. Optom. **8**:117-127, 1933; (f) Clinical Developments and Observations in the Correction of Aniseikonia, ibid. **9**:31-43, 1935.

2. Jackson, Edward: The Importance of Aniseikonia, Tr. Sect. Ophth., A. M. A., 1936, p. 27-48. Friedenwald, J. S.: Diagnosis and Treatment of Anisophoria, Arch. Ophth. **15**:283-307 (Feb.) 1936. Ludvigh, E. J.: Aniseikonia, Am. J. Ophth. **19**:292-301, 1936.

reported by these patients included functional disturbances, such as asthenopia, photophobia and disability in reading; local ocular disturbances, such as aching and burning of the eyes and blurring of print, and neurologic disturbances, such as headaches, nervousness and general fatigue. A large percentage of the patients obtained remarkable relief from their ocular discomfort when their aniseikonia had been corrected. As is to be expected in such a group, there were also many patients who did not obtain relief. The fact, however, that so many of the patients had a beneficial reaction from the treatment indicated the clinical value of investigating this anomaly as a possible cause of some of the disturbances mentioned.

A statistical study of the patients treated at The Eye Clinic was carried out during the past six years, during which time 829 patients

TABLE 1.—*Classification of Two Hundred and Eighty-Three Clinical Cases in Which Aniseikonia Had Been Corrected and in Which Definite Relief from Ocular Disturbances Was Reported*

	Number of Cases	Percentage
(a) Changes in the refractive corrections made at the time of correcting the aniseikonia		
Refractive correction not changed...................................	38	13
Refractive correction changed less than 0.50 D.	65	23
Refractive correction changed more than 0.50 D.	68	24
No classification as to refractive changes..........................	112	40
(b) Magnitude of refractive errors		
Refractive errors of both eyes less than 0.50 D.	31	11
Refractive errors of both eyes between 0.50 and 1.00 D.	32	11
Emmetropia	14	5
(c) Refractive condition of the two eyes		
Bilateral emmetropia	14	5
Isometropia	74	26
Anisometropia of 0.25 to 0.50 D.	76	27
Anisometropia of 0.50 D. or more...................................	119	42
(d) Prisms removed when the correction for aniseikonia was given........	19	6

were examined for aniseikonia. Of this number, 625 were given glasses to correct their aniseikonia, and, so far as is known, 500 of these are now wearing such corrections. Between November 1934 and May 1936, reports were received from 425 patients to whom letters had been sent requesting information as to the relief obtained from the correction for their aniseikonia. Two hundred and eighty-three patients reported definite relief from the particular symptoms of which they had complained. This number of cases in which the aniseikonia had been corrected successfully seemed to be sufficient to warrant a practical statistical study of the relationships between aniseikonia and ametropia.

STATISTICAL RESULTS

The tabulation of the responses to inquiries sent to the 500 patients who are wearing corrections for aniseikonia is given in the table and

shows that 283, or 57 per cent, have obtained definite relief from certain symptoms of ocular discomfort. These figures agree closely with those found at other clinics[3] where aniseikonia is measured and corrected.

TABLE 2.—*Graphic Representation of the Data of Table 1* *

Clinical cases in which aniseikonia had been corrected and in which definite relief from ocular disturbances was reported

(*a*) Changes in the refractive corrections made at the time of correcting the aniseikonia

    Refractive correction not changed

    Refractive correction changed less than 0.50 D.

    Refractive correction changed more than 0.50 D.

    No classification as to refractive changes

(*b*) Magnitude of refractive errors

    Refractive errors of both eyes less than 0.50 D.

    Refractive errors of both eyes between 0.50 and 1.00 D.

    Emmetropia

(*c*) Refractive condition of the two eyes

    Bilateral emmetropia

    Isometropia

    Anisometropia of 0.25 to 0.50 D.

    Anisometropia of 0.50 D. or more

(*d*) Prisms removed when the correction for aniseikonia was given

% 10 20 30 40 50 60 70 80 90 100

* The number of cases is given opposite each classification, and the corresponding percentage value can be read by means of the scale at the bottom of the figure.

COMMENT

It has been suggested that the relief obtained by persons with aniseikonia may have been due to a change in the correction for ametropia. Data of the table and figure show that for 38 of the 283 patients successfully treated no change in the correction for ametropia was made in the prescription which also corrected the aniseikonia. Since

3. Footnotes 1 *c, d, e* and *f*.

small refractive changes, of less than 0.5 D., are generally held to be insignificant, we may add to this group 65 other patients, for whom the change did not exceed 0.37 D. Thus, one finds 103 patients for whom no significant change in the correction for ametropia was made in the prescription which also corrected the aniseikonia. This combined group represents 36 per cent of the 283 patients who reported relief. We may, then, safely conclude that the relief from ocular discomfort obtained by these patients resulted, for the most part, from the correction of the aniseikonia.

There were, however, 68 patients for whom a radical change in the refractive correction seemed necessary or advisable. An attempt was made in many of the cases to determine whether relief could be obtained by means of the new refractive correction alone. In some of the cases the financial or physical condition of the patient did not permit such controlled experimentation.

The remaining 112 patients, or 40 per cent of the total number of 283, could not be classified in this manner for one of the following reasons: (1) The glasses had been discarded because no relief had been obtained; (2) neither glasses nor any record of previous prescriptions was available; (3) glasses had never been worn before.

At a recent conference a group of ophthalmologists expressed considerable interest in the degree of refractive error present in persons in whom aniseikonia has been demonstrated. Of the 283 patients who obtained relief by glasses for correction of aniseikonia, 63, or 22 per cent, were found to have a refractive error of 1 D. or less. This group may be subdivided as follows: 31 patients having a refractive error of 0.50 D. or less and 32 patients having a refractive error varying from 0.62 to 1.00 D.

While persons who have the condition true emmetropia are relatively rare, 11 patients with this condition have been examined and treated at The Eye Clinic. These patients, however, had definite symptoms of ocular discomfort, and although the cause of this ocular discomfort could not be found, they had been told repeatedly that the basis of their trouble was not ocular. For the reasons already stated, we are justified in adding to this group 3 patients whose refractive errors did not exceed 0.25 D. and who obtained complete relief by means of the correction for their aniseikonia without the use of a refractive correction. This gives a total of 14 patients, which represents 5 per cent of those reported on.

Just as there are persons with emmetropia who have aniseikonia, there are many more for whom the correction for ametropia is exactly the same for both eyes. Correction for aniseikonia has been included in the prescription for 74 patients with isometropia. This

fact shows that the correction of the refractive error in these cases is not the cause of the aniseikonia.

That aniseikonia is due to the correction of anisometropia is only partially true. Cases do occur in which, apparently, the aniseikonic magnification compensates for the aniseikonia produced by the correction for anisometropia. In the group of 283 patients under discussion, 195, or 69 per cent, were anisometropic; that is, there was a difference of refraction between the two eyes of 0.25 D. or more in one or both of the principal meridians. In order to clarify the relationship between anisometropia and aniseikonia a statistical treatment was made of the data for 119 patients who showed anisometropia of 0.50 D. or more in one or both of the principal meridians. In each case the refractive difference between the two eyes in each of the eikonic meridians (i. e., the meridian in which the aniseikonia was measured) was computed, and this difference was then compared with the difference in size actually measured in the clinic. Thus, the correction of astigmatism occupies an important place in such analysis. The results of the comparisons of the meridians may be summarized as follows:

1. The total number of meridians in which comparisons were made was 184.

2. The number of meridians in which the aniseikonia measured was in the same direction as that which might be expected from the correction of the anisometropia in those meridians was 136, or 74 per cent of the total number.

3. The number of meridians in which the aniseikonia measured was in the *opposite* direction from that which might be expected from the correction of the anisometropia in those meridians was 28, or 15 per cent of the total number.

4. The number of meridians in which either no aniseikonia was present, though anisometropia was corrected, or aniseikonia was measured, though no anisometropia existed, was 20, or 11 per cent of the total number.

This summary shows, first, that in the majority of the meridians, 74 per cent, the differences in size were in the direction that might be expected from the differences in power. This suggests that a part of the aniseikonia found had been introduced by the correction of the astigmatic ametropia. Second, in 15 per cent of the meridians the differences in size were in the direction opposite that which might be expected from the differences in power. This fact indicates that the basic aniseikonia was actually reduced by the correction of the astigmatic error. Finally, in 11 per cent of the meridians either aniseikonia existed without anisometropia or anisometropia existed without anisei-

of the ametropic condition.

Further statistical analysis, by correlation of the data obtained in these 119 cases, gave a coefficient of correlation of 0.6 [4] between the differences in size and the differences in power. Such a correlation shows that, although there is a general tendency for the correction of anisometropia to contribute to the amount of aniseikonia, no one to one relationship exists; that in certain cases of anisometropia there may be no aniseikonia at all, while in others the aniseikonia may be partially or totally reduced rather than increased by the correction for aniso-metropia. Furthermore, as has already been mentioned, since 74 patients with isometropia and 14 patients with bilateral emmetropia showed significant amounts of aniseikonia, and since there were 20 meridians in which there was anisometropia without aniseikonia, or vice versa, it is evident that aniseikonia may occur independently of the refractive error. It can be definitely stated, therefore, that on the basis of the ametropia present in any specific case no arbitrary deductions can be made as to the probable amount of aniseikonia.

Finally, more definite relief from ocular disturbance was obtained in 19 cases of heterophoria when the correction of aniseikonia was substituted for the correction of heterophoria by prisms. This fact shows that the heterophoria did not play a part in the etiology of the subjective disorders in these cases.

Five clinical cases have been selected which illustrate the various combinations of refractive conditions that may occur in cases of aniseikonia.

TYPICAL CASES

Case 1.—Mr. H. G. R., a business man aged 53, was wearing the following correction on Dec. 10, 1929: a —3.75 D. sph. $\bigcirc$ + 4.25 D. cyl., ax. 10 $\bigcirc$ 1.00 D. prism base in for the right eye and a —3.00 D. sph. $\bigcirc$ + 4.00 D. cyl., ax. 165 $\bigcirc$ 1.00 D. prism base in for the left, with the addition of a + 1.75 D. sph. for each eye.

For six years the patient had suffered from almost constant headaches in the frontal and occipital regions, which were frequently accompanied by gastric symptoms. His eyes became excessively tired during near work, and he experienced pain and a burning sensation in the eyeballs, as well as photophobia. He was unable to do near work except for short intervals not exceeding a total of an hour a day. The patient was not robust, but no systemic conditions had been found that would account for his distress. His refractionist had been unable to relieve the trouble, and he was referred to The Eye Clinic.

The same refraction was found, and tests for heterophoria showed exophoria of 1 prism diopter for distance and exophoria of 6 prism diopters for near vision. The examination for aniseikonia showed that the right ocular image was

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4. The prediction value of a coefficient of correlation of 0.6 is only 20 per cent.

smaller than the left by 1.75 per cent in the horizontal meridian. The correction for aniseikonia was given in a fit-over form, the original refractive correction without prisms being used. The patient returned five months later, on May 20, 1930, and reported that generally his eyes had given him less trouble than previously. There was still discomfort, but the periods of severe headaches were farther apart. He was reexamined in November 1930 and in April 1931. Although gradual improvement was reported at each visit, the basic prescription was not changed, but the correction for size was modified slightly.

In September 1934, after a careful recheck of the findings, the original refractive prescription was again given, together with a further, though slight, change in the correction for aniseikonia. The prescription was as follows: a —3.75 D. sph. ⊃ + 4.25 D. cyl., ax. 10 ⊃ 1.50 per cent meridional magnification, ax. 90 for the right eye and a —3.00 D. sph. ⊃ + 4.00 D. cyl., ax. 162 ⊃ 0.75 per cent meridional magnification, ax. 180 for the left. This correction was made up in permanent form as bifocal spectacles, with an addition of + 2.25 D. for each eye. In July 1935 he reported that these glasses were satisfactory and that it was possible for him to do more reading, since his symptoms had gradually disappeared.

CASE 2.—Mrs. A. W., a housewife aged 35, first visited the clinic on July 31, 1934. For the past fifteen to eighteen years she had suffered rather constantly from ocular fatigue, photophobia and car sickness and had occasional frontal headaches. The symptoms became much worse after a half-hour of reading or viewing motion pictures. Her ocular symptoms were accompanied by general physical fatigue and increase of nervous tension, for which repeated medical examinations had failed to disclose any organic basis.

Examination with the ophthalmo-eikonometer verified the emmetropic condition indicated by objective and subjective tests of refraction. There was esophoria of 0.25 prism diopter for distance and exophoria of 2 prism diopters for near vision. The left ocular image was found to be smaller than the right by 0.75 per cent in the vertical meridian and by 1.50 per cent in the horizontal meridian. The correction for this condition was given in a temporary prescription on July 31, 1934.

The patient reported partial relief after a short time, and as she continued to wear the glasses the symptoms of long standing gradually disappeared. In June 1935 she reported that she could read or do close work for from seven to eight hours a day without becoming nervous or tired and that the photophobia had completely disappeared. The car sickness had diminished so that the nausea had ceased, but long trips still caused general fatigue and a tired feeling around the right eye. On the basis of this report, glasses in a permanent form were furnished. In August 1935 she stated that her glasses continued to be a great source of comfort to her and that there was apparent improvement in her health.

At present, nearly two years after the first glasses for correction of the aniseikonia were given, she still has comfortable use of her eyes.

CASE 3.—Mrs. A. R., a housewife aged 63, first came to The Eye Clinic in April 1934 and complained of excessive lacrimation, tiring of the eyes during near work, and headaches in the frontal and occipital regions, which had been more or less constant throughout her whole life. When she read she became nauseated within a few minutes. When riding in a train or car she had to keep her eyes closed or refrain from looking out of the windows. She was afraid to cross streets because of extreme dizziness. Her physician had put her on a strict diet because of chronic biliousness.

On May 10 she was given the following prescription: a + 2.75 D. sph. ⊃ + 1.00 cyl., ax. 5 for the right eye and a + 4.75 D. sph. ⊃ + 1.75 D. cyl., ax. 5 ⊃ 1.25 per cent overall magnification for the left. One notes that the correction for the left eye is 2 D. stronger than that for the right and that the magnification added to the left lens for the correction of the aniseikonia is opposite to what one might expect. In a few weeks she reported marked improvement. She stated that she had not had any headaches, that she was not as nervous as formerly and could cross streets without fear and that she could read and sew without trouble. The car sickness and biliousness had disappeared, and her diet was not limited.

Two years later, in May 1936, she reported that she had fewer headaches than previously and these were rarely accompanied by nausea, that she could read comfortably and no longer rode with her eyes closed or feared to cross the street.

CASE 4.—Mrs. B., a weaver in a mill, aged 33, was referred to The Eye Clinic by an ophthalmologist because his refractive findings confirmed the ones of the practitioner who had examined her the year before and because the amount of error did not seem sufficient to explain the severe symptoms of which she complained. A general physical examination was found to give negative results.

Although her general health was fairly good, she had suffered from migraine headaches for sixteen years. These occurred weekly and lasted from one to three days. The attacks started with severe pain in the right eye, accompanied by lacrimation, fatigue and photophobia, followed by nausea and vomiting which made it necessary for her to go to bed until a day after the symptoms had subsided. Reading and motion pictures tired the eyes and usually brought on a headache. Riding in trains or cars always brought on a severe attack. She was unable to do steady work, as she was able to be up only two or three days a week. With reference to the family history, it was found that her mother had had similar headaches for many years.

External examination of the eyes and examination of the fundi gave negative results. The following refraction was obtained: with a —0.75 D. sph. ⊃ —1.50 D. cyl., ax. 90 vision of the right eye was 6/10, and with a 0.00 D. sph. ⊃ —0.25 D. cyl., ax. 180 vision of the left eye was 6/6. There were a fairly normal muscle balance for 20 feet (6 meters), exophoria of 2 prism diopters for this distance and exophoria of 7 prism diopters for near vision.

Examination with the ophthalmo-eikonometer in December 1934 revealed a moderately marked difference in size in the images of the eyes, which was in such a direction and of such an amount as to equal the aniseikonia produced by the correction for the anisometropia. A prescription was given which incorporated with the refractive correction already given a magnification of the right image of 2 per cent in the horizontal meridian.

The patient wore these glasses for a month, during which time there was no recurrence of the attacks of migraine. Her eyes were more comfortable, and she experienced no visual difficulties when viewing motion pictures.

On May 31, 1935, she reported that her eyes were fine and that she was feeling much better generally. She drove 300 miles (483 kilometers) and said that it was the first time in years that she had been able to take an automobile trip without experiencing a "sick headache." She has had no recurrence of the migraine, and her eyes do not feel tired at night after a day's work in the mills as they did previously. During the past year she has returned to The Eye Clinic on three occasions to have the glasses adjusted, and each time she has reported that there has been no recurrence of the so-called migraine.

CASE 5.—Mrs. S., a housewife aged 63, was first seen on Nov. 9, 1933. Her chief complaint was ocular fatigue. She stated that her eyes became tired during near work, and that such work was followed by headaches in the occipital region and vertigo, which were diagnosed by her internist as a mild form of migraine. She had to limit her reading to periods of fifteen minutes.

Ophthalmoscopic examination failed to reveal any pathologic findings except a small atrophic spot at the inner margin of the disk of the left eye. Her refraction was as follows: a + 2.00 D. sph. ⊃ + 0.50 D. cyl., ax. 115 for the right eye and' a + 2.50 D. sph. for the left, with the addition of a + 2.50 D. sph. for near vision. With this correction she exhibited esophoria of 16 prism diopters for distant vision and esophoria of 6 prism diopters for near vision. She was given a temporary prescription, which incorporated a lens for the correction of the aniseikonia before the right eye with a prism of 2 D. base out before both eyes. This arrangement did not prove to be satisfactory; so the lens for the correction of the aniseikonia was discarded and the prism power increased to 5 D. before each eye. The latter combination was worn from May to Dec. 8, 1934.

The patient reported that there was considerable relief and that she was able to work from four to five hours a day, although she still complained of an ache in the right eye and in the occipital region.

At this time she was seen by Dr. Bielschowsky. His examination revealed esophoria of 13 arc degrees with the eyes in the primary position, which remained the same on dextroversion; in that position it was combined with negative vertical divergence of 2 arc degrees, but it decreased slightly on levoversion and still more on supraversion and infraversion. For near vision he found esophoria of from 6 to 7 arc degrees.

It was decided to remove the prisms and to try the effect of only lenses for the correction of the aniseikonia for near vision. The prescription was made up as follows: a + 4.50 D. sph. ⊃ + 0.50 D. cyl., ax. 90 ⊃ 1.50 per cent meridional magnification, ax. 180 for the right eye and a + 4.00 D. sph. ⊃ + 0.50 cyl., ax. 90 ⊃ 1.75 per cent meridional magnification, ax. 110 for the left.

These glasses were used for all close work, and on Feb. 1, 1935, the patient reported: "These last lenses [for correction of the aniseikonia] are much more efficient than the preceding ones [prisms base out], and I do not see why they should not be called very successful. I did not expect a pair of new eyes, and I am far more comfortable than I ever expected to be. I can work five or six hours a day on difficult and demanding work."

The patient was examined again by Dr. Bielschowsky on February 15; he found, with the eyes in the primary position, esophoria of from 12 to 13 degrees which remained nearly the same on dextroversion and levoversion and decreased on supraversion and infraversion. For near vision, with the refractive lenses alone, the patient exhibited esophoria of from 10 to 11 arc degrees, and with the lenses for the correction of the aniseikonia added, esophoria of only from 7 to 8 arc degrees.

A third examination was made by Dr. Bielschowsky on April 9. Esophoria of a similar variation was found, being of from 14 to 15 arc degrees for distant vision and of from 6 to 9 arc degrees for near vision.

This patient presented a condition of relatively high esophoria both for distant and for near vision, which remained practically the same throughout all the tests. Correction of the esophoria with only prisms base out brought partial relief, but correction of the aniseikonia *without recourse to prisms at all* brought full and permanent relief.

SUMMARY

The data presented in this report may be summarized as follows:

Of the 829 patients examined for aniseikonia at The Eye Clinic, 625 were given prescriptions for the correction of their aniseikonia, and 500 of these were wearing the corrections at the time this study was begun.

Of the 500 patients to whom questionnaires were sent, 425 replied relative to the efficacy of the glasses for the correction of their aniseikonia.

Two hundred and eighty-three patients reported definite relief from ocular and general symptoms following the correction of their aniseikonia.

For 103, or about one third, of these patients no significant change was made in the refractive condition; hence it may be concluded that the relief obtained was a result of the correction of the aniseikonia.

For 68 patients, or about one fourth of the entire number, a significant change was made in the refraction, which leaves equivocal the results obtained in this group.

The cases of the 14 patients with emmetropia and the 74 with isometropia in whom significant amounts of aniseikonia were corrected show that aniseikonia may exist *independently* of ametropia.

Since 195, or 69 per cent, of the patients with aniseikonia were anisometropic, the presence of anisometropia suggests the possibility of concomitant aniseikonia.

The coefficient of correlation of 0.6 between aniseikonia and anisometropia indicates that, although a correction for ametropia may be a partial cause of aniseikonia in the general run of patients, there are also many persons in whom either there is no relation between the two or the correction of the ametropia may tend to reduce rather than to increase the basic aniseikonia.

The fact that the patients with emmetropia and isometropia showed aniseikonia of a sufficient amount, together with the fact that a coefficient of correlation of 0.6 was found between aniseikonia and anisometropia, show that on the basis of the ametropia no arbitrary deductions as to the possible amount of aniseikonia which may be present in any specific case are allowable.

When the correction for the aniseikonia was substituted for the prisms which had corrected marked heterophoria in 19 cases, definite relief from symptoms resulted.

CONCLUSIONS

The clinical results so far obtained justify the following conclusions: First, one of the important steps in obtaining efficient binocular vision is the correction of aniseikonia; second, in the cases of all patients

with functional disturbances of the eyes, including those who have failed to obtain relief by the correction of their refractive errors or motor anomalies, a clinical investigation for aniseikonia should be made; third, on the basis of the ametropia presented by any specific patient no arbitrary deductions as to the probable amount of aniseikonia can be made, and, finally, since anisometropia and aniseikonia are often concomitant, although not commensurate (or highly correlated), all patients with anisometropia should be examined for aniseikonia.

LIGHT ADAPTATION AT THE MACULA

AN EXAMPLE OF ITS INDUSTRIAL IMPORTANCE

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PHILADELPHIA

This paper is the result of work done in the laboratory for ophthalmology at the Wills Hospital for the National Brotherhood of Railway Firemen and Enginemen. The study was made in connection with the petition of this body before the Interstate Commerce Commission to compel the installation of automatic stokers on all coal-burning locomotives. This petition applied to all railroad runs whereon it is necessary for the firemen to be on the lookout for track signals, and to all occasions when they (the firemen) are specifically charged with the correct calling of such signals, as to both noting them on time and calling them correctly.

There were some other important factors connected with this petition before the Interstate Commerce Commission, such as economy of operation and the usual fatigue connected with firing by hand, but this paper has to do only with the factor just mentioned.

Firemen claimed before the commission that the effects on their eyes of the illumination from the fire box made it impossible for them to see the signals and to call them properly. This claim was based on their practical experiences and had only one important and serious point; i. e., after firing they were blinded for a certain length of time, during which it was impossible for them to see and to call the illuminated railroad signals.

Considering the speed of a railroad train when it is traveling with the throttle wide open and the distance which it covers in the time during which they were temporarily blinded, the firemen were aware of the dangers connected with such unguarded running. Disciplinary measures were being applied so frequently to the firemen because of their apparent disregard of signals, discovered when checking their observations against the observations of the driver of the locomotive, that they felt they were compelled to petition the Interstate Commerce Commission as they did.

In the final hearing various methods of protection, such as filters, goggles and visors, were discussed, but each had definite and serious defects. In the final analysis, protection from the illumination was the cause, and any protection from the illumination hindered the railroad man in his subsequent lookout for signals.

The laboratory work which had to be done in connection with this problem was: (1) simulation of the illumination of a fire box, (2) adaptation of persons to this illumination, (3) the simulation of a three point signal light with its illumination corrected for various distances and (4) determination of the effects of the illumination of the fire box on an observer's reaction time for finding and for calling correctly the simulated three point position light signal. The illumination of this signal was computed and adjusted for distances of 100, 300, 500 and 1,000 yards (91, 273, 455 and 910 meters). Basic fire box illumination consisted of from 4,000 to 5,000 foot-candles per square inch (6.3 sq. cm.) of the surface exposed, which is the usual illumination of a Mogul type of locomotive fire box, at from 36 to 38 inches (91 to 96 cm.) from the frame of the door of the fire box. Actual measurements with a Weston photometer at 6 feet (1.8 meters) from the fire box gave a reading of 500 foot-candles per square centimeter. The actual foot-candle power of the standard railroad signal light at 1 foot (30 cm.) from the housing was 50 foot-candles per square centimeter. The illumination at the housing for colored signal lights, considered as the standard for the green light, was 36 foot-candles per square centimeter. The available distance in the laboratory for simulating the three point position light was 30 feet (9 meters) from the observer to the position light.

The distances at which a fireman was exposed to the illumination of the fire box were carefully estimated under working conditions on the railroad run; the minimum was found to be 38 inches (96 cm.) and the maximum 6 feet (1.8 meters). Further, in estimating working conditions it was necessary to determine the lengths of time during which a fireman was exposed to this illumination. The distances at which the firemen were exposed were measured on a locomotive with a meter rule and the lengths of exposure were determined with a stopwatch. Six series of reaction times (generally accepted by railroad men) resulted from this.

Series A consisted of five exposures of 3 seconds each, with a 3 second interval, that is, an equivalent of the time required to open the fire box, throw in coal, close the door and repeat this process five times in ½ minute. Series B consisted of an unbroken exposure of 5 seconds; series C, of an unbroken exposure of 10 seconds; series D, of an unbroken exposure of 15 seconds, and series E, of an unbroken exposure of 25 seconds.

The test conditions in series B, C, D and E were connected with firing and with the trimming of the fire bed.

The test in series F consisted of ten exposures of 3 seconds each, with 3 second intervals between exposures. This was also connected with dressing the fire bed for obtaining maximum fire efficiency.

It was manifestly unnecessary to work with 500 foot-candles per square centimeter at 6 feet. A rheostatically controlled light was therefore used for the light adaptation, the illumination being reduced by the square of the working distance to a working distance 3 or 4 inches (7.5 or 10 cm.) from the eye. The foot-candle power of the position lights was similarly rheostatically controlled and was also reduced by the square of the distance to the proper foot-candle power, even to the third or fourth decimal place, depending on the distance simulated. The advantage of light adaptation at 8 to 10 cm. from the eye with a proportionately decreased illumination over utilization of a proportionately increased illumination at the actual working distance of 36 to 38 cm. from the eye is evident.

Under circumstances of dark adaptation the retina is normally able to perceive light in decreasing degrees of illumination even to an arbitrarily chosen normal of 0.00001 lambert. In light adaptation the reverse is true. Practically speaking, when the macula has been adapted or subjected to an illumination of a greater degree, exhaustion of the macular biochemical elements makes it impossible for the person to see lesser degrees of illumination until the effects of the greater illumination have disappeared. The macula, while sensitive so far as direct vision is concerned, becomes exhausted rapidly under increasing degrees of exposure to light. The sensitivity of the extramacular region increases by over one thousand times more than the macula itself when any problem in difference in light is present; but the fireman must use the macula for correct identification even though the signal was first perceived by an extramacular region.

The observers used in this study were firemen employed by the railroad. All were tested for visual acuity, for normal fields of vision and for a normal threshold for light after dark adaptation. The fundi were examined in each instance, and no one was used as an observer who did not have normal visual acuity and a normal fundus. Repeated readings were taken until each subject gave results which were consistent for that person. Subjects who gave high readings constantly (there were several such persons) were excused, and their readings were not utilized in the final recapitulation. The averages reported were the lowest recorded. Repeated tests were made on the subjects

finally selected, and their readings were presented in the hearing before the Interstate Commerce Commission. Table 1 gives the averages for the series:

TABLE 1.—*Averages of Reaction Time for the Series*

Series	Average Time, Seconds	No. of Observers
A	26	10
B	24	10
C	25	9
D	38	12
E	48	10
F	56	13

Though not included in the report of the findings, readings were found for series A, C and F as high as 2 minutes; for series E up to 9 minutes, and for series F and D of 2 minutes and 10 seconds and of 2 minutes and 40 seconds, respectively. The higher readings were not as frequent as the averaged lowest readings, but they appeared with disconcerting frequency. They are given in table 2.

TABLE 2.—*Averages of High Reaction Time for the Series*

Series	Average Time, Seconds	No. of Observers
A	60	3
B	68	3
C	64	4
D	90	1
E	98	3
F	102	1

All the observers were first subjected to dark adaptation to determine the presence of a normal curve for dark adaptation. Each observer was then taken separately and subjected to the light adaptation of the series under investigation. The light in the tests of light adaptation was controlled by a switch, and the duration of exposure was controlled with a stop-watch. The position light was placed 30 feet from the observer at varying degrees from his left side and at varying heights to simulate the actual working conditions. It was placed in the position of stop, go or caution. As soon as the light was switched off, the interval of time until the subject saw the position light and called it correctly was noted. His efficiency in perceiving colored lights was then tested. Tests with colored lights showed a delay in the perception of red light over yellow-white light of a minimum of 10 per cent; in the case of green light there was a decrease of 20 per cent, and in the case of blue light, a decrease of 100 per cent. It is interesting that this proved the usual considered theory in regard to the transmissibility of colors that when the relative efficiency in red, green and blue illumination is considered, matters are definitely in favor of red. Rice, Uhthoff and others have long held that the degree

of visual efficiency as regards visual acuity is highest in the perception of white light, next highest in the perception of red light, next highest in that of green light and least in that of blue light. For instance, in series E, in calling the red light the least interval was 1 minute and 10 seconds, while the greatest was 5 minutes. For the green light the least interval was 21 seconds and the greatest 5 minutes and 42 seconds. For the blue light the least interval was over 2 minutes and the greatest 7 minutes. It must not be forgotten that the subjects were all experienced railroaders.

Some interesting conclusions can be drawn from these findings. Their accuracy is incontestable. The physical set-up of the test was checked and rechecked, and if any errors were made they were in favor, constantly, of higher readings. This was proved by readings made in the cabs of locomotives; in almost every instance the intervals of time there determined when checked against the intervals determined in the laboratory were longer than the latter.

The effect of both night driving and day driving was included in the study. The findings were essentially the same in daylight driving as in night driving, undoubtedly because of the great degree of fire box illumination. Also, though nonilluminated semiphore signals replace, in part, the illuminated position lights, the central scotoma is present regardless of whether the signal viewed is a day-time semiphore or a night-time position light. Repeatedly the subjects described their finding of the approximate position of the signal light by their indirect vision and its subsequent disappearance within the field of the scotoma when they directed their macular vision thereon. When they were under the effects of the longer exposures a greater number of mistakes occurred in calling the signals, *after the signal light was seen.*

The increasing curve for the interval in series B, C, D and E is interesting. Five times the constant exposure did not give a proportionately longer interval. The interval is actually only twice as long. On the other hand, the ratio which exists between series A and series F is upset inversely. In series A the five exposures to light of 3 seconds each totaled 15 seconds of exposure and 15 seconds of darkness, while in series F there was 30 seconds of exposure with intervals of 30 seconds of darkness between the exposures. In series A the average was a 26 second interval, while in series F, in which the test condition was a condition frequently met in firing, the interval was slightly greater than twice that amount. It is interesting to compare these effects of light adaptation on visual acuity and on speed of discrimination and the disproportionate effects of increasing the illumination with certain established facts in regard to the variation of intensity of illumination. The summation curve is not a regular one; instead it constantly

approaches or tends to approach a vertical line of maximum efficiency, regardless of increasing illumination.

For instance, Ferree and Rand showed that in using illuminations ranging from 0.001 to 20 foot-candles there was a gain in visual acuity, with an increase from 0.001 to 0.01 foot-candle, or 489 per cent. With an increase from 0.01 to 1 foot-candle there was a gain of 63.7 per cent. With an increase from 1 to 5 foot-candles a gain of 30 per cent occurred, and with an increase from 5 to 25 foot-candles a gain of only 8.2 per cent occurred. Rice's figures were somewhat differently expressed but illustrate the same point. According to his findings, with an intensity of 0.015 foot-candle visual acuity was 0.386. With an intensity of one hundred times this amount, that is, 1.5 foot-candles, there was visual acuity of 1.464, while with an intensity of one thousand times the amount visual acuity was increased to only 2.087. With three times the last amount, or three thousand times more than the original intensity, visual acuity was increased to only 2.2—a total gain over the original efficiency of but 17.54 per cent. In regard to the effect of the intensity of light on the speed of discrimination, with an illumination of 0.4 foot-candle, according to Ferree and Rand, the speed of discrimination was 3.525 seconds. With an illumination of ten times this amount, or 4 foot-candles, the speed of discrimination was 8.654 seconds. With an illumination of forty-eight times this amount the speed of discrimination was 23.81 seconds, which is less than eight times the speed obtained with the lower degrees of illumination. In regard to the effect of the intensity of the light on the speed of adjusting the eye for clear seeing at different distances, with an illumination of 2.04 foot-candles there was a normal reading of 0.411 second. With an illumination five times this, that is, 2 foot-candles, the reading was 0.568 second, while with 12 foot-candles, that is, an illumination forty-eight times the amount of the original, there was a further increase of only 0.316 second. These findings are quoted only to show the effects which varying degrees of illumination have on the macular region in that increasing the illumination does not increase the efficiency of discrimination proportionately. They are concurred in not in actuality (for confirmation of these findings was not the problem of our laboratory) but decidedly in substance, for we discovered similar relationships with the very high and the very low degrees of illumination.

The findings were reported before the Interstate Commerce Commission, and testimony was given thereon. The decision of the Interstate Commerce Commission is not especially relevant to the presentation of this work. The work is presented simply as an application of laboratory methods in an attempt to solve a serious industrial problem which can have a far reaching effect both in safeguarding the lives of railroad

passengers and in making necessary or unnecessary the expenditure of huge sums of money by the railroads for equipment not considered by some a definite necessity.

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DISCUSSION

DR. FRANCIS HEED ADLER: I should like to call attention to the fact that each determination was made immediately after exposure to the artificial fire box illumination. It would not be likely that under actual conditions the signals would come each time after the fireman had looked into the fire box. Unless one had data showing how often signals are presented and how often the fire box must be opened one could not tell what the chances were of the moment after exposure to fire box illumination and that at which signals come coinciding, and since the scotoma lasts a relatively short time I do not believe that this is the main reason why so many signals are missed.

DR. H. MAXWELL LANGDON: Dr. Spaeth said one thing that I thought was a slight exaggeration: that a train may run uncontrolled for a short time. Is that correct?

DR. EDMUND B. SPAETH: The average firing conditions on the short runs between Philadelphia and Atlantic City, for instance, were determined. On the longer runs, where there are miles without signal lights, these conditions, of course, would not apply. In answer to Dr. Langdon: My statement implying that a train may run uncontrolled for a short time may be a slight exaggeration, and the explanation should be made that there are many times, as stated at the final hearing, when signals are visible only to the fireman and not to the engineer.

ASTROCYTOMA (TRUE GLIOMA) OF THE RETINA

REPORT OF A CASE

JOHN M. McLEAN, M.D.
BALTIMORE

The purpose of this paper is to report an apparently unique retinal tumor. So far as can be found, no similar tumor has been reported in the literature. This tumor is a true glioma of the retina—an astrocytoma.

The term glioma of the retina was first used by Virchow to describe tumors now known as retinoblastoma. It was later pointed out that such tumors were not glial in nature and differed essentially from the true gliomas of the brain to which they had been likened. Wintersteiner,[1] following Flexner's[2] lead, introduced the term neuro-epithelioma retinae, and Verhoeff[3] suggested the better name retinoblastoma, by which the tumor is now known in this country. With the exception of a few French authors,[4] ophthalmologists have abandoned the term glioma of the retina, generally believing such an entity does not exist. However Verhoeff in 1932,[5] in reviewing the subject of glioma of the optic nerve, called attention to the fact that there was true glial tissue in the retina and that sooner or later a true glioma of the retina might occur. The finding of the tumor reported here bears out this prophecy.

Glial tissue has been noted before in primary retinal tumors, having occasionally been demonstrated in the rare tumor of the pars ciliaris retinae first designated as teratoneuroma[6] and later renamed diktyoma.

1. Wintersteiner, H.: Das neuroëpithelioma Retinae, Vienna, Franz Deuticke, 1897.

2. Flexner, S.: A Peculiar Glioma (Neuroepithelioma?) of the Retina, Bull. Johns Hopkins Hosp. 2:115, 1891.

3. Verhoeff, F. H.: Tr. Am. Ophth. Soc. 24:38, 1926.

4. Morax, V.: Pathologie oculaire, Paris, Félix Alcan, 1929. Redslob, E.: L'histogénèse des tumeurs de la rétine, Bull. Soc. d'opht. de Paris, March 1934, p. 123.

5. Verhoeff, F. H.: Tumors of the Optic Nerve, in Penfield, Wilder: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1027.

6. Verhoeff, F. H.: A Rare Tumor Arising from the Pars Ciliaris Retinae (Terato-Neuroma), Tr. Am. Ophth. Soc. 10:351, 1904.

7. Fuchs, E.: Wucherungen und Geschwülste des Ciliarepithels, Arch. f. Ophth. 68:534, 1908.

In these instances it has been present as a supporting framework and not as a part of the primary tumor. More recently a similar glial network has been noted by Grinker[8] and illustrated in detail in an article by Urra[9] as it appeared in the much more common retinoblastoma. In these tumors also the glial framework, like the nutrient vascular supply, is strictly a secondary proliferation and independent of the true tumor.

Von Hippel's disease and Lindau's disease[10] were once believed to be closely related to primary gliomatous growths in the retina. However, Lindau[11] recently has shown this is a misconception, these tumors being true angioblastomas with only slight and secondary glial proliferation.

Van der Hoeve[12] has described a peculiar cystic tumor sometimes found in the fundus of patients with tuberous sclerosis. The growth is typically cystic, edematous and degenerative.[13] Histologic evidence of low grade inflammation is common, while ruptures of the cysts, local implantation metastases to distant areas of the retina and free cysts in the vitreous seem to be the rule rather than the exception. The site of origin is usually, if not always, in the nerve head. In the histopathologic description of such a tumor[14] are mentioned areas with mere swelling of the elements of the nerve fiber layer and similar areas where there are also large cells said to be undifferentiated neurocytes. All the patients with a retinal lesion of this type also had typical tuberous sclerosis of the central nervous system, usually accompanied by the equally typical adenoma sebaceum and often by the less common visceral lesions.[15] There are certain points of similarity between the tumor reported here and this lesion, but there are also sufficient differences to clearly differentiate it from the phakomatosis just mentioned.

8. Grinker, R. R.: Tumors of the Retina, in Penfield, Wilder: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1041.

9. Urra, F.: Ueber die feine Gewebsstruktur des Glioms der Netzhaut, Arch. f. Ophth. 112:133, 1923.

10. (a) Meller, J.: Ueber das Wesen der sogenannten Hippelschen Netzhauterkrankung, Arch. f. Ophth. 85:255, 1913. (b) von Hippel, E.: Ueber diffuse Gliose der Netzhaut und ihre Beziehungen zu der Angiomatosis retinae, ibid. 95:173, 1918.
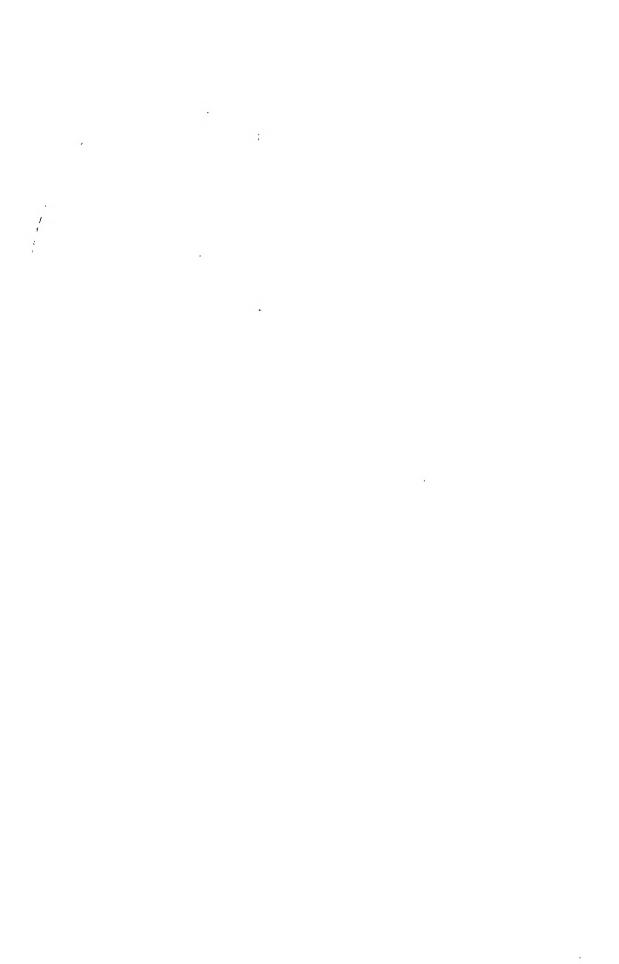
11. Lindau, A.: Zur Frage der Angiomatosis retinae und ihre Hirnkomplikationen, Acta ophth. 4:193, 1927.

12. van der Hoeve, J.: Eye Symptoms in Tuberose Sclerosis of the Brain, Tr. Ophth. Soc. U. Kingdom 40:329, 1920; Augengeschwülste bei der tuberösen Hirnsklerose, Arch. f. Ophth. 105:880, 1921.

13. van der Hoeve, J.: Eye Diseases in Tuberose Sclerosis of the Brain, Tr. Ophth. Soc. U. Kingdom 43:534, 1923.

14. van der Hoeve, J.: Augenschwülste bei der tuberösen Hirnsklerose und verwandten Krankheiten, Arch. f. Ophth. 111:1, 1923.

15. van der Hoeve, J.: Eye Symptoms in Phakomatosis, Tr. Ophth. Soc. U. Kingdom 52:380, 1932.
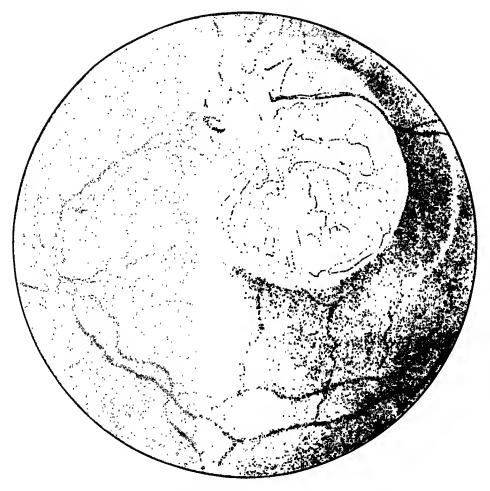
Fig. 1.—Ophthalmoscopic appearance of the tumor.

The only other apparent glial tumefactions of the retina are the massive gliosis, described by von Hippel[10b] and by Friedenwald,[16] and the glial proliferation found either in massive, long-standing, inflammatory disease of the retina or as the end-result of degenerative processes.

The tumor reported in this paper is an isolated primary tumor of the retina which appears to be entirely distinct from all the aforementioned forms hitherto described.

REPORT OF CASE

History—Mrs. F. S., a 23 year old white woman, was first seen in the Wilmer Ophthalmological Institute on March 7, 1936. Two days before she had suddenly noticed that the vision of her left eye was very poor. There was no previous ocular history of any sort.

The family history was entirely irrelevant.

The past history was essentially unimportant, with no record of serious illnesses or injuries. There was a history of dysuria and frequency and urgency of urination of several months' duration. The only operation was a tonsillectomy at the age of 21.

General and Special Examinations.—The general physical and special neurologic examinations gave entirely negative results. Gynecological examination showed bilateral pyelitis, and cultures of urine obtained by urethral catheterization showed a moderate growth of Staphylococcus albus from each kidney and growth of Bacillus alkaligenes from the left kidney. Treatment was instituted later for this condition.

The blood counts, the blood chemistry and the cytologic picture were all normal. The Wassermann reaction of the blood was negative.

Examination of the Eye.—External examination showed both eyes to be normal, with normal pupils, tension and extra-ocular movements. Vision of the right eye was 20/15. Vision of the left eye was 20/70 and was not improved by refraction.

Ophthalmoscopically, the right eye showed clear media and a normal fundus. In the left eye the media were clear and the disk was normal. There was a mass in the left macular region, about 3 disk diameters in size, projecting forward about 8 D. and sharply circumscribed. It was smooth and whitish, with many vessels running over the surface. No extension could be made out, and the rest of the fundus was not abnormal in any way (fig. 1).

The visual field for form and that for color were normal in the right eye. The peripheral limits of the fields were normal in the left eye, but there was a large absolute scotoma extending nasally and somewhat down from the point of fixation for about 20 degrees.

The slit lamp examination gave entirely negative results for each eye.

A diagnosis of intra-ocular tumor in the left eye was made, and the eye was enucleated on March 9, with the patient under the influence of general anesthesia. The postoperative course was uneventful.

Pathologic Examination.—Macroscopic Observations: The globe was fixed in Bouin's solution and then opened in a frontal plant in the region of the ora

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16. Friedenwald, J. S.: Massive Gliosis of the Retina, in Crisp, W. H., and Finnoff, W. C.: Contributions to Ophthalmic Science Dedicated to Edward Jackson, Menasha, Wis., George Banta Publishing Co., 1927, p. 23.

serrata. The anterior half was normal. In the posterior half neither the disk nor the vitreous appeared unusual. The retina and the choroid were in place. In the posterior pole, 4 or 5 mm. directly temporal from the disk, was a pale, almost white, smoothly rounded tumor about 5 mm. in diameter and 2 mm. high. No other lesions could be made out.
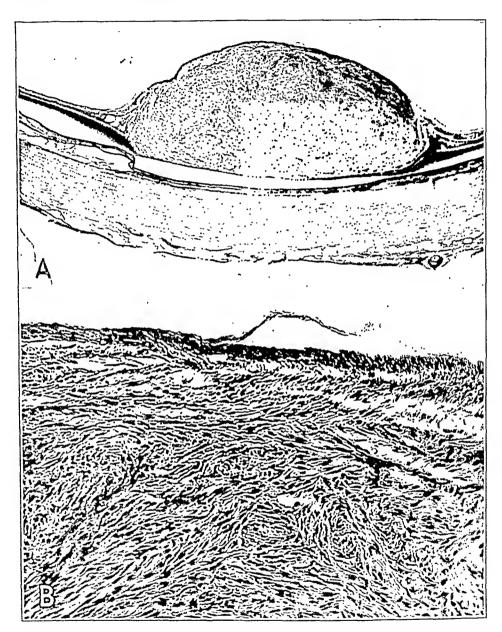
Fig. 2.—*A*, photomicrograph showing displacement of the normal retinal elements by tumor cells. *B*, photomicrograph showing the inner limiting membrane intact over the tumor.

Microscopic Observations: Horizontal sections were made and studied with the following stains: the hematoxylin and eosin stain, the Verhoeff-Van Gieson stain for elastic tissue, Mallory's aniline blue and phosphotungstic acid stains, the trichrome stain, silver impregnations and the Wilder stain for reticulum.

Except for the tumor the eye was entirely normal. The tumor was confined to the retina, supplanting and displacing the normal retinal elements (fig. 2 A). The inner limiting membrane was intact over the tumor, which did not invade the
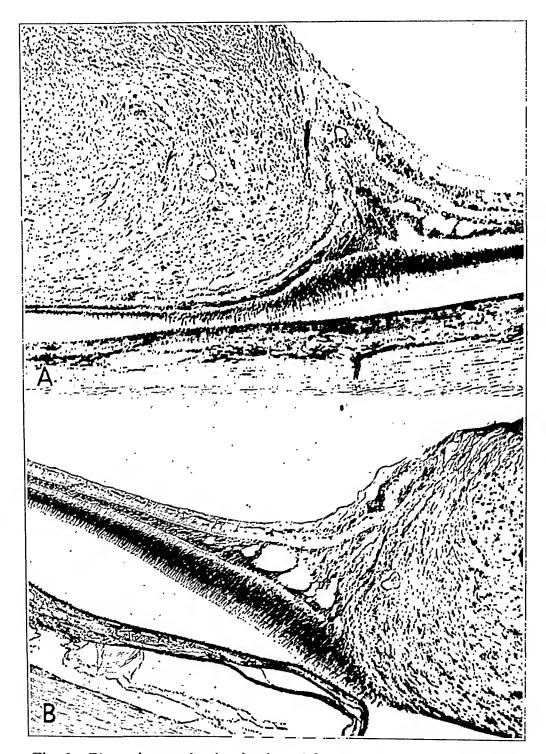
Fig. 3.—Photomicrographs showing lateral fading out of the tumor into normal retinal elements.

vitreous (fig. 2 B). The rods and cones were for the most part preserved, but they were markedly thinned under the center of the tumor, where the pigment epithelium appeared compressed (fig. 4 A). Bruch's membrane was intact, and

there was no invasion of the choroid. Laterally the tumor faded out into normal retinal elements (fig. 3). While there was a certain amount of either invasion or displacement of the outer layers, the tumor was predominantly in the inner layers of the retina and appeared to have arisen there.
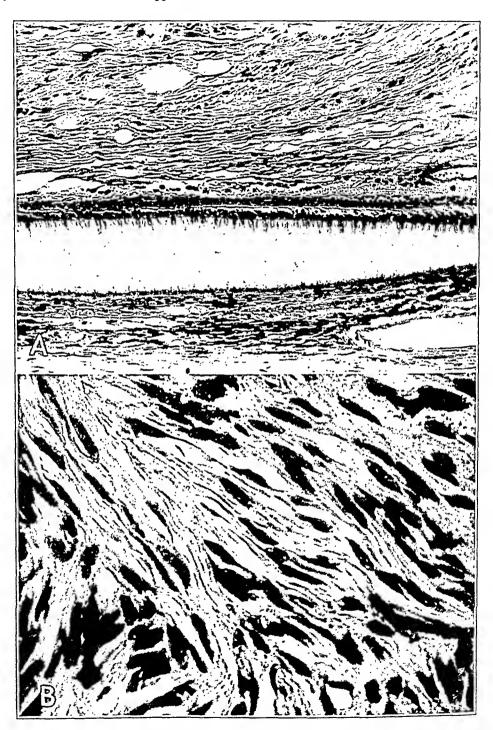
Fig. 4.—*A*, photomicrograph showing thinning out of the rods and cones under the center of the tumor, where the pigment epthelium appears compressed. *B*, photomicrograph showing long spindle cells with single, medium-sized, ovoid nuclei containing scattered masses of chromatin, a moderate amount of cytoplasm and long fibrillary processes.

The tumor was composed for the greater part of long spindle cells which had single medium-sized ovoid nuclei containing scattered masses of chromatin, a moderate amount of cytoplasm and long fibrillary processes (fig. 4 B). Differential stains showed that these were neuroglial, not connective, tissue. There were also scattered clumps of large irregular cells with abundant eosinophilic cytoplasm and round or oval nuclei about twice the size of the nuclei of the ganglion cells in the normal retina. These stained fairly deeply, but many small granules and a few larger clear areas were made out within them. In many places the cytoplasm was drawn out into fibrillary axon-like processes. Occasional multinucleated cells were seen, and in some of the large cells the nucleus seemed to be distorted and displaced as if by swelling of the cytoplasm. Mitoses were excessively rare. There were many thin-walled blood vessels, but there was only a suggestion of astrocytic "sucker feet" about them. There was no communication with the choroid, and the blood supply seemed to be entirely from the retinal system.

COMMENT

Study of the cellular morphologic features of this tumor, together with its reaction to differential staining, leads to the conclusion that it must be classed as an astrocytoma arising from true gial tissue. The existence of fibrillary astrocytes in the inner layers of the retina has been recognized for years.[17] These are similar to the astrocytic glial elements of the central nervous system, where astrocytoma is the type of glioma most frequently encountered.[18] Perhaps the appearance of origin in the inner layers of the retina is significant in respect to the normal location of retinal astrocytes.

The age of the patient is of some interest. She was well beyond the usual age for retinoblastoma and in the age group for the remarkably rare retinal sarcoma[19] which originates in connective tissue of the retinal blood vessels. However, astrocytoma of the brain is usually found in the second and third decades of life[18]—in the age group of this patient.

The unusual retinal tumors in the phakomatosis of Bourneville (tuberous sclerosis) are the only lesions at all similar to the tumor in this case. That rare disease shows definite familial characteristics, whereas this patient's family history was irrelevant. Phakomatosis also involves the central nervous system primarily, with associated cutaneous and visceral lesions and occasional tumors of the eye. There was no evidence in this patient's history or in the results of the examinations

17. Greeff, R.: Die mikroskopische Anatomie der Netzhaut, in von Graefe, T., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1900, vol. 2, p. 74.

18. Bailey, P., and Cushing, H.: Tumors of the Glioma Group, Philadelphia, J. B. Lippincott Company, 1926.

19. Schick, F.: Das Perithelium der Netzhautzentralgefässe, Arch. f. Ophth. 81:328, 1912. Elschnig, A.: Zur Kenntnis der primären Retinatumoren, ibid. 87:370, 1914.

to suggest any such general or neurologic involvement. Vogt[20] reported a typical retinal nodule of tuberous sclerosis, the article being illustrated by an excellent colored drawing, and quite properly likened it to a mulberry. The ophthalmoscopic picture in the case reported by me contrasts with the ophthalmoscopic picture in Vogt's case. The cystic, degenerative, edematous character of the lesions in Vogt's case, together with their frequent rupture, reimplantation, and free nodules in the vitreous, further differentiate them from the tumor in the case I have just reported, in which all such features are conspicuously absent. Nevertheless, just as the microscopic picture in tuberous sclerosis of the brain bears some resemblance to astrocytoma of the brain, so a high power photomicrograph of one of the lesions in Vogt's case might show a picture resembling that seen in a high power photomicrograph of the retinal tumor reported here. This likeness cannot be entirely overlooked, but it seems impossible to group these conditions together in the face of so many points of differentiation. Perhaps with the possible accumulation of other cases of astrocytoma of the retina and more cases of tuberous sclerosis with associated retinal lesions a better understanding of the points of similarity and contrast may be had.

No definite opinion can be given on the relative malignancy of this tumor. Cytologically, it appears to be relatively benign. It is not particularly invasive. There are neither abundant mitoses nor other evidences of rapid· growth. Metaplasia is not excessive. There is no information as to the time of onset of the growth, for the patient found the visual defect only accidentally, the other eye being normal. Since astrocytoma is the least malignant glioma of the brain,[18] by analogy this tumor should be of low malignancy. Since there are no evidences of metastases and enucleation has given thorough and complete removal of the growth, the prognosis should be good.

20. Vogt, A.: Seltenen Maulbeertumor der Retina bei tuberösen Hirnsklerose, Ztschr. f. Augenh. **84**:18, 1934.

TREATMENT OF CAUSTIC BURNS OF THE EYE

WILLIAM B. HUBBARD, M.D.

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Chemical burns of the eye are of frequent occurrence. Cowan and Sinclair[1] in their recent review of blindness in Pennsylvania found that blindness was more often due to chemical burns than to detached retina. Detached retina has received much attention, while the study of chemical burns of the eye has been neglected.

In this paper I desire to discuss briefly the emergency treatment of burns of the eye caused by soluble acid and alkali and to present new experimental studies.

In advising treatment of caustic burns experimental work must be considered of paramount importance. The factors, such as (a) the time that has elapsed before initial treatment, (b) the amount of the caustic causing the burn, (c) the type of the caustic, (d) the strength of the caustic and (e) the type of treatment, are so variable that one cannot outline the proper treatment from clinical observation alone.

In a previous experimental study[2] on caustic burns it was found that, regardless of the concentration of a chemical or of the interval of time before treatment, the proper treatment was to irrigate the eye with water rather than to attempt neutralization. With neutralization chemical reaction with heat was apparent. Mechanical removal of the chemical was the most important factor. This was shown in the experiments with phenol, which is not soluble in water. The tremendous importance of the time that had elapsed before first aid was given was demonstrated. It was further shown that after a certain time of exposure for any concentration irreparable damage had been done. Some of the newer texts are advising treatment along the lines mentioned. A great many continue, however, to advise neutralization. The advocates of neutralization should have incontestable experimental proof of its desirability, as advising neutralization is practically the same as advising against any emergency treatment. Before a proper neutralizing solution could be obtained all the damage would have been done.

1. Cowan, Alfred, and Sinclair, S. M.: Causes of Blindness in Pennsylvania from Medical and Social Aspects, J. A. M. A. **107**:757-760 (Sept. 5) 1936. Sinclair, S. M.: Personal communication to the author, Oct. 21, 1936.

2. Cosgrove, K. W., and Hubbard, W. B.: Acid and Alkali Burns of the Eye, Ann. Surg. **87**:89-94 (Jan.) 1928.
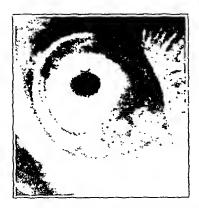
Fig. 1.—Normal rabbit eye. Note the transparent cornea, with the iris and the pupil clearly seen, and the normal lids. The nictitating membrane is prominent.
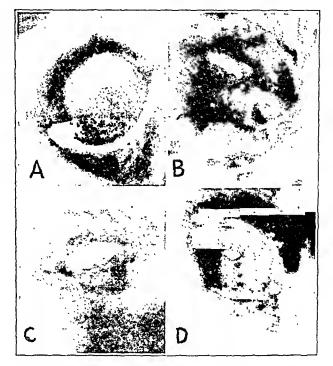
Fig. 2.—*A*, rabbit eye twenty-two days after exposure for thirty seconds to a 32 per cent solution of sulfuric acid. The eye was treated with vigorous irrigation with water. Note the rather clear cornea and the good condition of the eyeball. *B*, rabbit eye twenty-two days after exposure for thirty seconds to a 32 per cent solution of sulfuric acid. The eye was treated with vigorous irrigation with a 2 per cent solution of sodium bicarbonate. Note the partially degenerated eyeball. A comparison of *A* and *B* shows that treatment of an acid burn by irrigation with water is more efficacious than treatment by irrigation with a weak alkali. *C*, rabbit eye twenty-two days after exposure for ten seconds to a 20 per cent solution of sodium hydroxide. The eye was treated with vigorous irrigation with water. Note the completely degenerated eyeball. *D*, rabbit eye twenty-two days after exposure for ten seconds to a 20 per cent solution of sodium hydroxide. The eye was treated with vigorous irrigation with a 2 per cent solution of acetic acid. Note the almost normal eye. A comparison of *C* and *D* shows that treatment of an alkali burn by irrigation with weak acid is more efficacious than treatment by irrigation with water alone.

NEW EXPERIMENTAL STUDIES

In the experiments with caustic burns of the eye reported in this paper the effect of irrigation with water was compared with the effect of irrigation with a weak neutralizing fluid. With this procedure harsh chemical reaction was avoided. The mechanical factor of removal was present in both cases.

The effect was studied when a 32 per cent solution of sulfuric acid and a 20 per cent solution of sodium hydroxide were instilled into the eyes of rabbits. Eleven drops of caustic were used. The sulfuric acid was allowed to act for thirty seconds and the sodium hydroxide for ten seconds. These solutions finally destroyed useful vision in the majority of cases.

In these experiments I decided to irrigate the right eye with tap water in all cases. The left eye after the sulfuric acid burn was irrigated with a 2 per cent solution of sodium bicarbonate and after a sodium hydroxide burn with a 2 per cent solution of acetic acid.

Every possible precaution was taken to perform the experiments with accuracy. Two experienced assistants aided. The caustics and irrigating solution were carefully measured, and the time was regulated exactly by a stop watch.

Between 80 and 100 per cent of the acid burns treated by irrigation with a weak alkali were definitely worse than the acid burn of the opposite eye treated by irrigation with water alone.

Seventy-five per cent of the alkali burns treated by irrigation with a weak acid were·definitely better than the alkali burn of the opposite eye treated by irrigation with water alone.

It is well known that acid proteinates are insoluble and that alkaline proteinates are soluble. The findings in these cases substantiate the theory that a soluble alkaline proteinate does not protect against further injury, although an insoluble acid proteinate does. These findings do not prove or disprove the value of irrigation, as all the eyes were irrigated in one way or another.

If sulfuric acid is a typical acid, an eye with an acid burn should be irrigated with water and not with a weak alkali. If sodium hydroxide is a typical alkali, an eye with an alkali burn should, when possible, be irrigated with a weak acid.

COMMENT

The findings in this group of eyes raise interesting points for additional experimental studies: 1. There are many more substances to be studied to determine the best emergency ocular treatment. 2. After-treatment of burns of the eye should be studied experimentally. Modified studies with tannic acid seem indicated. The present studies suggest that acidity of the conjunctival sac is advantageous after any type of burn. 3. The treatment of caustic burns of the skin and the gastrointestinal tract should receive further attention experimentally. In

regard to the latter, it appears incorrect to prescribe a weak alkali for a person poisoned by an acid. This disagrees with treatment advised in the present literature.

SUMMARY

Acid burns of the eye should be treated immediately by irrigation with water and not with a neutralizing fluid—neutralizing substance in the water is harmful.

Alkali burns of the eye should be treated immediately by irrigation with a weakly acid solution. If this is not practicable, they should be irrigated with water and a weak acid should be instilled as soon as possible.

It is believed from these findings that further studies should be made of other parts of the body, such as the gastro-intestinal tract.

Further experimental studies of caustic burns of the eye are indicated.

PRIMARY CARCINOMA OF THE LACRIMAL SAC

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MINNEAPOLIS

Owing to the rarity of primary carcinoma of the lacrimal sac, the following case warrants recording.

REPORT OF A CASE

History.—E. J., a man aged 78, consulted me in June 1927 with regard to a mass over the right lacrimal sac. This growth was first noticed about two months prior to the time of the examination and had slowly increased in size. His only complaint was of epiphora.

Examination.—The vision of each eye was normal. Over the region of the right lacrimal sac was a firm, smooth, round mass approximately from 1 to 1.5 cm. in diameter. The skin was not red and was freely movable. Neither tears nor pus could be expressed from the puncta. A diagnosis of mucocele was made and removal advised.

Operation.—With the part under local anesthesia, the lacrimal sac was excised after the method of Meller. The anterior wall was much thickened and firmer than one would expect from an inflammatory condition. The sac contained a small amount of mucus. The wound healed per primam intentionem, and the patient returned home in three days. The appearance of the wall of the sac was suggestive of a new growth.

Microscopic Examination.—Dr. W. A. O'Brien, of the University of Minnesota, reported as follows: "The specimen was a small white tumor from eye. Sections showed nonpigmented cords and masses of epithelium resembling squamous epithelium. The diagnosis was carcinoma."

Second Operation and Course.—In August, eight weeks later, the patient returned, and a hard fibrous mass, 1.5 cm. in diameter, was removed by a second operation.

The patient was seen again in June 1929. There was a thickened elevated area 2.5 by 3 mm. on the inferior nasal portion of the cornea near the limbus. Two months later, in August, this area had spread to the limbus. This growth had involved the tissue beneath the conjunctiva, and there was slight limitation of motion of the eye. In May 1930 the patient was referred to Dr. Laura Lane for radium treatment. A pack containing 200 millicuries of radon was used at three distances, 1, 2 and 3 cm., with three different filters. The total dose amounted to 800 millicurie hours. On June 9, 600 millicurie hours was applied in a pack to the eyelid. When the patient was seen in March 1931 there was marked exophthalmos resulting from the extension of the growth backward. The cornea was cloudy and vision was reduced to counting fingers at 2 feet (61 cm.).

Third Operation.—In March 1931 exenteration of the right orbit was done, with the patient under nitrous oxide anesthesia. The eyelids were not removed. The postorbital space was found to be filled with a hard mass of tissue about 3 by 1 cm. in size.

Pathologic Report.—Dr. Charles Drake's report on the tissue was as follows: "Sections from the hard portion showed the structure to be made up of irregular masses of epithelium growing in all directions. Many mitotic figures were present. The microscopic diagnosis was carcinoma."
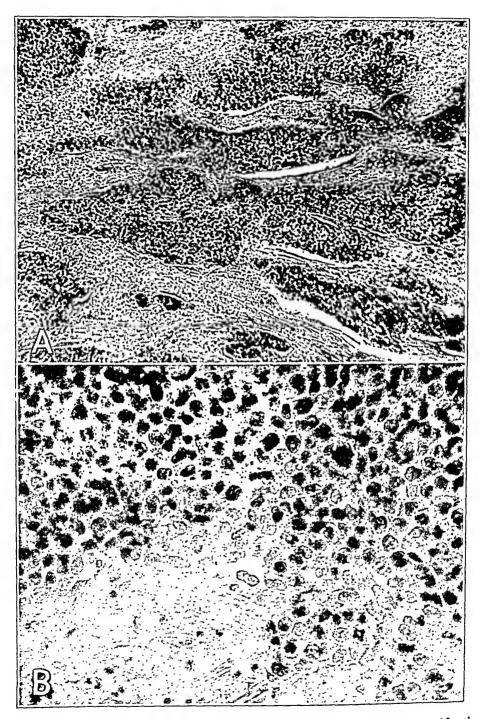
Fig. 1 (Spratt).—Carcinoma of the lacrimal sac. *A*, low magnification; *B*, high magnification.

Subsequent History.—The orbit filled in by granulation, and the lids became adherent to the scar tissue. The patient was seen again in April 1933, two years after the exenteration. There was no recurrence of the growth in the orbit.

There was, however, extensive metastasis to the submaxillary lymph nodes. He was referred to the University Hospitals for radium and roentgen treatment. Radon was implanted in the nodes on May 15 by Dr. Randall, who used a total of 28 millicuries in thirty-one gold implants. The thickness of the wall of gold was 0.3 mm. The total dose amounted to 3,690 millicurie hours. High voltage roentgen therapy was given to the right side of the face and neck over an area measuring 13 by 18 cm. on May 16 and 18. The factors were: 200 kilovolts, a 0.5 mm. copper filter plus a 4 mm. aluminum filter, a target-skin distance of 60 cm., 30 milliamperes, and a half value layer of 1 mm. of copper. The intensity used amounted to 33 roentgens per minute, and the total dose was 750 r. This therapy was followed by complete disappearance of the swelling. The patient died in July 1935 at the age of 86, eight years after the first appearance of the growth, four years after the exenteration of the orbit and two years after the radium treatment of the involved glands. There was no recurrence in the glands. The cause of death was coronary thrombosis.
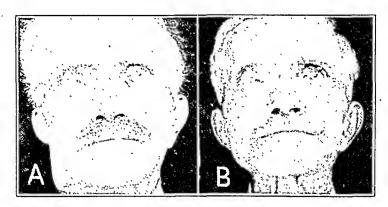
Fig. 2 (Spratt).—Enlarged glands at the angle of the jaw. *A,* appearance of the patient two years after exenteration of the orbit; *B,* appearance two years after the implantation of radium and roentgen treatment and four years after exenteration of the orbit.

COMMENT

Synopsis of Cases.—Parsons[1] has described many types of tumor of the lacrimal gland but made no mention of a new growth of the lacrimal sac. Angiosarcoma, angiomyxosarcoma, cystoma, endothelioma, lymphoma, lymphosarcoma, fibrosarcoma, plasmasarcoma, malignant papilloma and carcinoma as primary tumors of the lacrimal sac have been reported.

A survey of the ophthalmic literature revealed sixteen additional cases of primary carcinoma of the lacrimal sac. In this group of seventeen cases, thirteen of the patients were males, and four were females. Thirteen were persons more than 40 years of age. One patient was aged 36; one was 25 years of age; one was a young woman of unknown age, and a fourth was a boy aged 18 (in this case the growth was a malignant papilloma).

1. Parsons, J. H.: Pathology of the Eye, New York, G. P. Putnam's Sons, 1904.

Synopsis of Seventeen Cases of Carcinoma of the Lacrinal Sac

Author	Year	Male	Female	Age, Years	Right	Left	Duration	Diagnosis	Recurrence	Cure	Unknown
Piccoli: Lavori d. clin. ocul. d. r. Univ. di Napoli 4:257-265, 1891-1896; abstr, Ann. di ottal. (supp.) 24:10, 1895-1896	1895	..	+	56	+		8 to 9 mo.	Carcinoma	In 3 to 4 mo.	....	..
Sgrosso [13]	1900	+	..	65	Not given		4 yr.	Epithelioma	....	Did not recur	..
Dalén: Beitr. z. Augenh., 1899, no. 41, pp. 1-9	1902	..	+	24		+	2 yr. after extirpation of sac	Carcinoma	In 4 mo.	....	..
Lafon: J. de méd. de Bordeaux 36:172, 1906	1906	+	..	48		+	10 mo.	Epithelioma (cylindric cell)	Unknown	....	+
Rollet [4]	1906	+	..	65	....		....	Epithelioma	Unknown	....	+
Blatt: Arch. Ophth. 39:53, 1910	1909	+	..	60	Not given		....	Carcinoma	Yes	....	..
Pasetti: Ann. di ottal. 42:55-64, 1913	1913	+	..	73	....		....	Carcinoma	Not known	....	+
Posey [7]	1921	+	..	74	....		....	Epithelioma (cylindric cell)	....	In 2 yr.	..
Strada and Urrets Zavalía [11]	1925	..	..	53	....		2 mo.	Flat cell carcinoma	Patient died 10 mo.	....	..
Bakker and Oudendal [12]	1926	..	+	Young woman	Not given		1 mo.	Basal cell carcinoma	+	In 18 mo.	..
Hilden [2]	1929	+	..	18	+		Several months	Malignant papilloma	In 3 mo. (2 operations)	In 2 yr.; 2 operations	..
Dupuy-Dutemps [3]	1929	..	..	47	+		4 to 5 mo. (papilloma removed at 22 yr. of age)	Papillary epithelioma	In 4 mo.	....	..
Muirhead [5]	1933	+	..	60	+		5 mo.	Transitional cell carcinoma	Unknown	....	+
Tennent [6]	1933	+	..	63	+		2 mo.	Cylindric cell carcinoma	Unknown	....	+
Urrets Zavalía and Obregón Oliva [11]	1935	+	..	36		+	1 yr.; epiphora	Epithelial origin	In 3 mo.	....	..
Urrets Zavalía and Obregón Oliva [11]	1936	+	..	47	....		1 yr.	Carcinomatous	Gland at angle of jaw	In 4 mo.; 2 operations	..
Spratt	1936	+	..	78	+		2 mo.	Carcinoma	Orbit and angle of jaw; second operation performed	In 4 yr.; 3 operations	..

In two of these cases the growth was diagnosed as malignant papilloma (Hilden's[2] case and Dupuy-Dutemps'[3] case). In the latter case the patient had a papilloma of the lacrimal sac removed at the age of 22 years. When she was 47 years of age a malignant papilloma appeared at the site of the original tumor. This recurred after removal. (It is a question whether this case should be included in the series.)

The original articles were consulted when available, as titles frequently are misleading. As an example, the three cases of cancer described by Rollet[4] consist of one of primary carcinoma, one of sarcoma (polypus) and one of secondary melanotic sarcoma. Several writers have described carcinoma of the lacrimal sac secondary to tumor of the lid or antrum.

In the English literature, Muirhead[5] and Tennent[6] have each described a single case. The only case reported in the American literature is that described by Posey[7] in 1921.

De Vincentiis[8] (1876) is credited as having been the first to describe a primary cancer of the lacrimal sac. His original article was not available, but according to Silvestri,[9] who reviewed the Italian literature, the tumor was described as a papillary fibroma. Maggi[10] reported it as a tumor of the lacrimal sac.

The normal lining of the lacrimal sac is composed of cylindric cells. Repeated irritation due to chronic inflammation may cause this membrane to become thickened and form several layers of cells which change to the squamous variety. Consequently, microscopic examination may show the carcinoma to be the cylindric, the transitional or the squamous cell type.

Diagnosis.—Since the onset is insidious, with subjective symptoms no different from those of an ordinary cystic condition of the lacrimal

2. Hilden, B.: Malignes Tranensackpapillom, zugleich ein Beispiel von Zellenmetaplasie in den Tranenwegen, Klin. Monatsbl. f. Augenh. 82:661-665, 1929.

3. Dupuy-Dutemps, L.: Epithéliome papillaire de la fosse nasale consécutif à un papillome du sac lacrymal extirpé 24 ans auparavant, Bull. et mém. Soc. franç. d'opht. 42:215-220, 1929.

4. Rollet: Trois cas de cancer non ulcéré du sac lacrimal, Arch. d'opht. 26: 337, 1906.

5. Muirhead, W. N.: Carcinoma of the Lacrimal Sac, Tr. Ophth. Soc. U. Kingdom 53:591-592, 1933.

6. Tennent, J. N.: Carcinoma of the Lacrymal Sac: Report of Two Cases, Tr. Ophth. Soc. U. Kingdom 53:93-101, 1933.

7. Posey, W. C.: Report of a Case of Primary Tubular Epithelioma of Lacrimal Sac, Tr. Am. Ophth. Soc. 19:205-208, 1921.

8. de Vincentiis, C.: Di un raro caso di fibroma papillare del sacco lagrimale, Movimento, Napoli 8:418-423, 1876; quoted by Silvestri[9] and Maggi.[10]

9. Silvestri, A.: Sarcoma del sacco lacrimale, Ann. di ottal. 26:452-459, 1897.

10. Maggi, F.: Contributo allo studio dei tumori primitivi del sacco lacrimale, Ann. di ottal. 35:789-798, 1906.

sac, in only one of the cases in this series was the growth correctly diagnosed as malignant prior to operation. The appearance on examination is that of a hard, round mass with freely movable skin. Stenosis of the lacrimal duct is generally present.

In the first stage a correct diagnosis of carcinoma of the lacrimal sac is impossible, as epiphora is the only symptom. In the second stage, that of swelling, differentiation between a tumor and a mucocele can be made. A tumor gives rise to epiphora owing to pressure of the thickened wall of the lacrimal sac. Pressure on the sac causes little or no regurgitation, and the duct is patent on irrigation and on probing. Signs of inflammation are absent. In cases of mucocele the duct is generally obstructed. Pressure on a mucocele causes regurgitation of fluid into the nose or from the puncta on diminution in size of the swelling. A hard mass with rapid growth and lack of diminution in size on pressure indicates a new growth. In the third, or final, stage, characterized by rapid growth of the hard mass, ulceration and enlargement of the preauricular and the maxillary lymph glands, the growth presents little difficulty in diagnosis. The case reported by Strada and Urrets Zavalía [11] was the only one in which the growth was diagnosed as a malignant tumor before operation; in the other cases the growth was considered a mucocele. The only case in which pain was reported was that of Strada and Urrets Zavalía.

Prognosis.—These tumors are malignant, and recurrence is the rule. In five of the seventeen cases the end-results were not given. In three cases the growth was reported as cured. Posey's [7] case was observed two years; Bakker and Oudendal's [12] was observed eighteen months, and for the third, that of Sgrosso, [13] no time of observance was given. Recurrence was noted in nine of the seventeen cases. Two of the patients were operated on a second time; in one no recurrence was noted at the end of four months (Urrets Zavalía and Obregón Oliva [14]), and in the other, at the end of two years (Hilder [2]).

A third patient was operated on three times (my patient), and no recurrence was noted locally over a four year period. It is not unlikely that had these patients been traced, recurrence would have been found in all. There is no record of any patient having been free during the five year period.

11. Strada, F., and Urrets Zavalía, A.: Contribución al estudio de las tumores malignos del saco lagrimal, Semana méd. 2:1100-1108 (Oct. 29) 1925.

12. Bakker, C., and Oudendal, A. J. F.: Een geval van basalecullincarcinom van den ductus nasolacrimalis bij een inlandsche vrouw, Nederl. tijdschr. v. geneesk. 2:2-6, 1926.

13. Sgrosso, P.: Epitelioma del sacco lagrimale, Ann. di ottal. 29:82-96, 1900.

14. Urrets Zavalía, A., and Obregón Oliva, R.: Sobre un caso de carcinoma primitivo del saco lagrimal, Arch. de oftal. de Buenos Aires 10:521-537, 1935.

Treatment.—All the patients were operated on (by extirpation of the sac with the tumor). Since the diagnosis of the malignant nature was made in only one case (that of Strada and Urrets Zavalía) prior to operation, removal was incomplete and recurrence was the rule.

After · operation, Dupuy-Dutemps,[3] Strada and Urrets Zavalía,[11] Posey,[7] Urrets Zavalía and Obregón Oliva [14] (two cases), Muirhead [5] and I used radium or roentgen rays, with poor results. Local recurrence followed in all cases except those of Posey,[7] Strada and Urrets Zavalía [11] and mine. It appears that radium and high voltage roentgen therapy with proper filtration offer hope for cure if used after extirpation of a malignant tumor of the lacrimal sac.

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Clinical Notes

AN ENUCLEATION COMPRESSOR

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The instrument described in this paper is used during enucleation. Either the large or the small end is inserted into Tenon's capsule immediately after the optic nerve has been severed. By making pressure in

The enucleation orbital compressor.

the apex of the orbit one can compress the bleeding vessels of the orbital apex. This pressure will immediately stop the bleeding.

This instrument is held tightly against the apex of the orbit while the sutures are inserted in Tenon's capsule. The handle is made 10 inches (25.4 cm.) long in order that the assistant's hand holding the instrument may be out of the way of the operator. The shaft of the handle is small (6 mm. in diameter), so that it does not interfere while the operator is inserting sutures in Tenon's capsule.

The tips of this instrument are olive shaped; the large end measures 14 mm. and the small 11 mm. The large end is useful for operations on adults and children, and the small end is useful for operations on children under the age of 5 years.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

TUBERCULOUS LESIONS OF THE UVEAL TRACT
A REVIEW OF THE LITERATURE

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AND
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PHILADELPHIA

GENERAL CONSIDERATIONS

It is extremely difficult to prove that in a suspected case of uveal tuberculosis the condition is actually due to the tubercle bacillus. The history of the patient, the appearance of the lesion and the clinical course are all one has to rely on in making a diagnosis of uveal tuberculosis. A focal reaction to tuberculin is generally conceded, in this country, to be too dangerous to justify its use, and even when positive does not afford absolute proof of the nature of the lesion. The patient's cutaneous sensitivity to tuberculoprotein is a diagnostic aid, the significance of which we shall discuss later on. The absence of other possible etiologic factors to explain the lesion is further suggestive evidence. The Schilling differential blood count has been reported by Bredeck[1] as of diagnostic value, taken in conjunction with the subcutaneous tuberculin test. Other inflammatory conditions may also cause a similar shift in the blood picture, so that the test is not specific, and a diagnosis of tuberculosis cannot be made on the basis of the blood picture alone. Vernes' test has been used with some success by Williamson-Noble.[2] The test is sometimes positive in the absence of tuberculosis if the patient has had fever twenty-four hours before the blood has been taken, and a negative test is therefore said to be of more value than a positive one.

In the last analysis, no lesion can be diagnosed with absolute certainty as tuberculous until the eye is removed and sectioned and the histologic picture of tubercle noted, or until inoculation of tissue into animals succeeds in producing tuberculosis. Some pathologists may even question the histologic picture of tubercle unless the bacilli can be demonstrated in the lesion. It must be admitted, therefore, that a clinical diagnosis of tuberculous uveitis must be made *ex cathedra*.

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1. Bredeck, quoted by Post, M. H.: J. Tennessee M. A. **29**:179, 1936.
2. Williamson-Noble, F. A.: Brit. M. J. **2**:907 (Nov. 7) 1936.

Statistics vary as to the incidence of tuberculosis of the uveal tract in different clinics. In Germany one clinic [3] has reported that in as many as 50 per cent of the cases of iritis observed by them the condition was tuberculous, while in this country Irons and Brown [4] estimated that in less than 5 per cent of the cases of iritis observed by them the condition was caused by the tubercle bacillus. Tuberculosis, in its various forms, is not correspondingly more prevalent in Germany than in the United States. The discrepancy in these figures merely indicates that German ophthalmologists make this *ex cathedra* diagnosis more frequently than ophthalmologists in this country. This is true not only of tuberculous iritis but also of tuberculosis of the rest of the uveal tract.

There are at least two reasons why this diversity of opinion exists. Much more attention has been given in this country to other focal infections than to tuberculosis as the probable cause of lesions of the uveal tract, and there is no typical picture of tuberculous uveitis by which one can recognize this condition. The typical picture of tuberculosis, wherever it occurs in the body, is that of discrete nodular lesions, i. e., tubercles. These may be miliary or may coalesce to form large conglomerate tubercles, but in any case the essential feature is that of a well circumscribed nodular lesion.

When nodules are found on the surface of the iris during the course of iritis, one naturally thinks of tuberculosis, together with syphilis and other diseases in which nodules occur. If the iritis appears in the form of a plastic exudate, with no nodular lesions present, many clinicians in this country are not willing to consider a diagnosis of tuberculosis. Such cases are usually studied carefully from the standpoint of focal infections, and little attention is given to other causes once a negative serologic test has ruled out syphilis.

Prior to the time of von Michel it was generally believed that the only tuberculous diseases of the eye were those associated with the formation of tubercles. Von Michel [5] was the first to recognize the fact that, in spite of the absence of any macroscopic tubercles, the histologic sections of the iris in some cases of iritis showed miliary lesions which could be identified as true tubercles.

In this country one of the first to call attention to the possibility of tuberculosis in such cases was Verhoeff, [6] who reported a case of

3. Gilbert, W., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 3.

4. Irons, E. E., and Brown, E. V. L.: Recurrence of Iritis as Influenced by the Removal of Infections, J. A. M. A. 87:1167 (Oct. 9) 1926.

5. von Michel, in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer. 1930, vol. 5, p. 38.

6. Verhoeff, F. H.: Acute Tuberculous Iritis: Microscopic Examination of Eye Showing This Condition, Tr. Sect. Ophth., A. M. A., 1930, p. 21.

iritis the chief characteristic of which was acute fibrinous exudation with no formation of nodules. Tuberculosis was not suspected until the eye was removed because of excessive pain. Microscopic examination revealed the presence of characteristic tubercles in the rest of the uveal tract.

In the same manner, discrete miliary nodules seen in the choroid immediately suggest the possibility of tuberculosis, whereas in the forms of choroiditis in which the lesions are diffuse and highly exudative the condition is not likely to remind one of the possibility of this disease.

In 1932 Eggston [7] stressed the fact that tuberculosis may not always occur in the body in the characteristic form of tubercles. He pointed out that many conditions, undoubtedly of a tuberculous nature, are to be considered the expression of an allergic response on the part of the body to tuberculoprotein circulating in the blood, i. e., serous pleurisy, the cutaneous tuberculids and various ocular lesions, in which neither tubercles nor the tubercle bacilli could be demonstrated.

This conception of allergic tuberculous lesions is based on considerable experimental evidence. An excellent résumé of the work on this subject has been given by Allan Woods.[8]

EXPERIMENTAL PATHOLOGY

When a tuberculous infection is established in the body certain reactions take place in both the cellular and the humoral mechanisms. Perhaps the first defensive mechanism which is called into play is the cellular, and its expression is the collection of a group of lymphocytic cells around the invading organisms. If the bacilli are viable, these cells persist and change their character to that of epithelioid cells. Long and Holley [9] have shown that the large mononuclear cells, which ultimately give rise to epithelioid cells, come either from the blood stream directly or from the walls of the new ingrowing blood vessels, and not from the local fixed tissue cells, at least in the case of the cornea. If the bacilli are dead, no epithelioid cells may be formed, and the invading mononuclear cells may disappear, probably going back to the blood stream from which they came. Assuming that the invasion of bacteria is sufficiently virulent, a fight now takes place between the epithelioid cells and the bacteria. Giant cells form from the epithelioid cells. Some of the bacteria are engulfed and destroyed, while some of the cells

7. Eggston, A. A.: The Use of Tuberculin in Diagnosis and Treatment in Ophthalmology, Arch. Ophth. 8:671 (Nov.) 1932.

8. Woods, Allan: Allergy and Immunity in Ophthalmology, Baltimore, The Johns Hopkins Press, 1933, p. 98.

9. Long, E. R., and Holley, S. W.: Am. J. Path. 9:337, 1933.

are killed and form cellular detritus in the form of caseating material. If the bacteria succeed in breaking down this primary wall of defense they get into the blood stream and are carried either by the blood vessels or by the local lymphatics to fresh sites, and the infection spreads.

At the same time that the cellular defensive mechanism is doing its part, certain less well known humoral defenses are being formed. The nature of the substances in the blood stream is not well known. Antitoxin apparently does not exist. On the other hand, complement-fixing antibodies and agglutinins have been reported by a number of authors.[10] Once a tuberculous infection is established in the body, it is found that that body is more resistant to a second experimental infection. This increase in resistance has been called immunity.

When a subject whose immunity to tuberculosis has been increased is given a further injection of tubercle bacilli, either alive or dead, he may show a prompt and acute reaction, which is due to the sensitization of his tissues to the products of disintegration of the bacilli which caused the primary infection. This is called an allergic reaction. The subject has become sensitive, or allergic, to tuberculoprotein, and when he is further exposed to tuberculoprotein the result is a violent inflammatory exudative reaction.

Allergy does not occur with every infection of tuberculosis. If the infection is massive, the body may not respond by becoming allergic but may succumb before this stage can be reached. Further, allergy diminishes in the moribund patient suffering from tuberculosis. One group of investigators have expressed the belief that the allergic reaction is responsible for the development of immunity and that the two processes are really one and the same.[11] A second group have maintained that immunity is due not to the allergic reaction but to some other unknown factor, while allergy is an independent sensitization of the tissue cells to tuberculoprotein.[12] This school believes that the formation of miliary tubercles in the body, as a manifestation of tuberculosis, results when the number and virulence of the invading bacilli are high and the immunity low. If the body is allergic at the same time the result of such an infection may not be generalized miliary tuberculosis but inflammatory lesions of an extensive exudative, even necrotizing, type. This will be referred to in considering the various forms of uveal tuberculosis.

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10. Kunz, E.: Arch. f. Augenh. **109**:709, 1936.

11. Vaughn, W. T.: J. Lab. & Clin. Med. **21**:629, 1936.

12. (a) Paretzky, M.: Am. Rev. Tuberc. **31**:499, 1935; **33**:370, 1936. (b) Friedenwald, J. S., and Dessoff, J.: Bull. Johns Hopkins Hosp. **57**:148, 1935.

The difference between allergy and immunity has been clearly expressed by Rich and McCordock.[13] They defined allergy as a change in the body as a result of which the cells are more extensively damaged or killed by a given amount of tuberculoprotein than are similar cells in a normal body. Immunity, on the other hand, consists in the ability of an infected animal to hold locally tubercle bacilli introduced into the body, i. e., to prevent the bacilli from spreading from the site at which they first lodge, and, further, to prevent them from multiplying as rapidly as they would in the normal body. According to these authors, the bacilli must be held or fixed locally in the immune tuberculous animal by means of some specific precipitin-like substance which does not interfere with the free movement of other particulate matter.

The experiments of Friedenwald[14] support the belief of Rich and McCordock, that allergy and immunity are two different processes, independent of each other. Friedenwald was able to desensitize tuberculous guinea-pigs by giving repeated doses of tuberculoprotein over a long period. These desensitized animals showed no diminution in their resistance to reinfection, as judged by the mortality of the disease, the spread of the infection and the progress of the local lesion. In all these respects the desensitized animals showed greater resistance to reinfection than did the nondesensitized animals. This indicates strongly that allergy in tuberculosis cannot be regarded as essential to immunity. These results have been confirmed by Selter and Weiland.[15]

CLINICAL ASPECTS OF TUBERCULOUS UVEITIS

It is evident from what has been said that lesions caused by the tubercle bacillus may vary in their appearance and course, depending on such factors as the number and virulence of the infecting organisms, the state of immunity of the infected body and the degree of allergy of that body to tuberculoprotein. It is of interest now to review the various forms of uveitis and to see whether they can be explained on the basis of these variable factors.

Tuberculosis of the uveal tract is the most frequent form of ocular tuberculosis. It is practically always secondary to a tuberculous lesion elsewhere in the body and develops only in the second stage of tuberculosis, i. e., when a tuberculous nodule breaks down and the bacilli get into the blood stream. Ocular tuberculosis represents a metastasis, therefore, of tuberculous bacillemia. Once they get into the blood stream the bacilli are carried to the uveal tract and deposited there. Rarely

13. Rich, A. R., and McCordock, H. A.: Bull. Johns Hopkins Hosp. **44**:273, 1929.

14. Friedenwald, J. S.: Tr. Am. Ophth. Soc. **30**:269, 1932.

15. Selter, H., and Weiland, P.: Ztschr. f. Tuberk. **74**:161, 1935.

they may reach the uveal tract by way of the lymphatics or by direct extension of a tuberculous lesion from contiguous structures. Even in tuberculous choroiditis secondary to tuberculous meningitis the infection occurs by way of the blood stream, as Tooke [16] has recently shown, and not by spreading down the meninges.

If the ocular metastasis occurs shortly after the primary infection, before the body has become highly allergic, one should find the development of discrete miliary tubercles with little inflammatory reaction. Actually, the cases of tuberculous iritis which one sees clinically, in which there are many nodules in the iris and little inflammatory reaction, occur in young persons. There is little flushing of the ciliary body or pain, and not many precipitates are present on the posterior surface of the cornea, but studded here and there in the stroma of the iris are discrete yellowish or gray nodules which often disappear in the course of a few days or weeks, leaving small areas of atrophy. Often these nodules are too small to be seen except with the aid of magnification such as is possible with the slit lamp; this should be used in every case of iritis and a careful search made for these miliary lesions. Although the larger nodules seem to have a predilection for the ciliary margin of the iris, they occur frequently near the pupillary edge, and it is here that the smaller ones are most frequently found. This form of tuberculous iritis occurs in young persons from 10 to 25 years of age. The subjects frequently have a history of tuberculous contacts. They sometimes show definite pulmonary lesions on roentgen examination but are not likely to have any marked sensitivity to tuberculoprotein.

In the posterior portion of the uveal tract this type of iritis is best represented by the miliary tubercules of the choroid occurring in children with tuberculous meningitis. These are the easiest types to diagnose as tuberculous because of the frankly nodular character of the lesions.

In the next decade one finds a type of iritis more highly inflammatory in character, but with little tendency to the formation of nodules. If nodules are present, they are likely to be fewer. There are considerable pain and some plastic exudate, and the iris quickly becomes bound down with posterior synechia. This condition is frequently associated with a corneal condition, such as sclerosing keratitis. In the posterior segment this inflammation is represented by large greenish gray lesions, usually single, in the extreme periphery of the choroid. The vitreous is so hazy that often the details of the fundus cannot be made out, but careful search in the periphery reveals the characteristic white or greenish color of the underlying lesion against the dark or red background. The patients, ranging in age from about 20 to 30 years, usually have

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16. Tooke, F. T.: Tr. Am. Ophth. Soc. **33**:201, 1935.

a high degree of cutaneous sensitivity to tuberculoprotein and rarely show any active lesions in the lungs. The roentgen rays commonly show old healed lesions at an apex and enlarged peribronchial lymph glands.

Patients from 30 to 50 years of age show the next most common type of tuberculous uveitis met with. In these the condition is seldom iritis alone; the ciliary body is equally involved. The lesions are distinctly highly exudative. The whole posterior surface of the cornea is covered with mutton fat deposits. There is little flushing of the ciliary body or pain, but the whole iris is firmly plastered down to the lens when the patients first come for examination because of failing vision. If the pupil can be dilated the vitreous is found to be full of opacities which prevent any view of the fundus. In these cases the condition is usually bilateral and is a frequent cause of blindness. If secondary glau-

TABLE 1.—*Characteristic Features of Tuberculous Iritis*

Age	Type of Lesion	General Tuberculosis	Allergy*
10 to 20	Nodular discrete tubercules; little inflammation or exudate	Occasionally active lesions	Very little
20 to 30	Acute plastic iritis; few, if any, nodules; highly inflammatory; moderate exudation	Seldom active but healed lesions	Moderate
30 to 50	Chronic iritis; marked exudate; little inflammation; mutton fat deposits; occasional large nodules	Healed lesions	Marked

* As will be explained in the section on the Mantoux test, allergy is a quantitative matter. In our own tests we considered a patient as moderately allergic if he reacted positively to 0.0001 mg. of purified protein derivative. If he did not have a positive reaction to 0.01 mg., he was not considered allergic.

coma does not occur, because of the occlusion of the pupil, the globe generally becomes phthisical. A large percentage of these cases are found in the colored race. Nearly all the patients have a high degree of allergy. Clinical and roentgen examination of the chest reveals about the same picture as in the previous type of uveitis.

There is no characteristic lesion of the posterior segment of the uveal tract which corresponds to this type of iridocyclitis. Many of the German writers include in this category the various forms of disseminated choroiditis seen in patients at this age. At present the evidence seems insufficient to classify them as such; the lesions are certainly not highly exudative, and the condition seems to us to be more a degenerative than an inflammatory process. Perhaps tuberculosis should be considered, but it would be reprehensible to try to provoke a focal reaction with tuberculin for diagnostic purposes.

The tables summarize the characteristic features of the various forms of iritis and choroiditis commonly seen.

RELATIONSHIP OF OCULAR TUBERCULOSIS TO GENERAL
TUBERCULOSIS

Recent statistics show that almost 100 per cent of the adult popula-
tion of the large cities of this country have had at some time during
their life an infection with the tubercle bacillus, and this infection
has been sufficient to give them a cutaneous sensitivity to tuberculo-
protein.[17]

This would seem to indicate that ocular tuberculosis should be fairly
common, especially among those with active pulmonary lesions, and
yet the reverse is true. It is generally recognized that patients with
ocular tuberculosis seldom have active pulmonary lesions and that ocular
tuberculosis is extremely rare in sanatoriums for patients with pulmo-
nary tuberculosis. Of a thousand patients with active pulmonary tuber-
culosis examined by Goldenburg and Fabricant,[18] in only three could
the ocular condition be considered as due to tuberculosis, and only

TABLE 2.—*Characteristic Features of Tuberculous Choroiditis*

Age	Type of Lesion	General Tuberculosis	Allergy
1 to 10	Miliary tubercles of choroid	Tuberculous meningitis	Very little
10 to 30	Inflammation of the anterior part of the uvea; single large exudative lesion; little inflammation; tuberculous periphlebitis	Healed lesions	Moderate or severe
30 to 50	Disseminated choroiditis included by many German writers	Healed lesions	Not stated in the literature; our own results did not indicate any constant degree of allergy

nineteen patients showed any pathologic changes in the posterior seg-
ment of the globe which could have been tuberculous. This paradox has
never been satisfactorily explained, but we believe it is no proof of the
claim made by these authors that tuberculosis of the eye must be much
less common than the reports in the literature indicate.

In clinics where many patients with suspected ocular tuberculosis
are seen the consensus is that the vast majority show healed pulmonary
lesions or lesions of the peribronchial lymph glands.[19]

Axenfeld and de la Camp were never able to demonstrate any active
lesions in the lungs of their patients by the roentgen rays, and these

17. Hetherington, H. W.; McPhedran, F. M.; Landis, H. R. M., and Opie,
E. L.: Tuberculosis in Medical and College Students, Arch. Int. Med. **48**:734
(Nov.) 1931; Further Study of Tuberculosis Among Medical and Other University
Students, ibid. **55**:709 (May) 1935.

18. Goldenburg, M., and Fabricant, N. D.: Eye in Tuberculous Patient, Tr.
Sect. Ophth., A. M. A., 1930, p. 135.

19. Werdenberg: Klin. Monatsbl. f. Augenh. **75**:545, 1925. Axenfeld, T.:
ibid. **85**:465, 1930.

negative findings were confirmed by Wendt.[20] Werdenberg, on the other hand, found no person among one hundred and thirty patients examined in whom the lungs did not show some pathologic changes. Ninety of these patients showed moderate involvement, and thirty showed advanced tuberculosis of the apexes or at the hilus. Gilbert[21] also claimed that in practically every case of tuberculous uveitis the roentgen rays reveal some evidence of pulmonary tuberculosis.

THE MANTOUX TEST

As has been indicated, accurate diagnosis of uveal tuberculosis is difficult. Among the most important of the diagnostic aids is the intracutaneous tuberculin test, or the Mantoux test. When properly performed it is of great help in establishing the presence of cutaneous sensitivity to tuberculoprotein.

In the proper performance of the test certain precautions should be observed and certain errors avoided. The recommendations are as follows:

Method.—The intradermal test is rapidly supplanting other tuberculin tests, because the dosage can be accurately controlled and in that way undesirable local, focal and constitutional reactions can be avoided. These are particularly apt to occur in subcutaneous and Calmette (ophthalmic) tests.

Materials.—Many kinds of tuberculin have been tried. A potent old tuberculin is very satisfactory, but because of the variability in strength of different batches efforts have been made to produce a stable uniform product. To this end a purified protein derivative of the tubercle bacillus has been produced and tested by Seibert, Aronson, Reichel, Clark and Long. This promises to supplant old tuberculin because it is potent and its strength varies little in different batches. Test dilutions should be made up fresh at least once a month and refrigerated.

Syringes.—Each syringe should be reserved for its own strength of old tuberculin or purified protein derivative, to avoid false positive reactions.

Dosage.—As a high percentage of the population, especially the urban population, have sensitivity of the skin to tuberculin in high concentration,[17] it would seem important to determine the degree of sensitivity rather than its mere presence.

To this end the following schedule is suggested: The initial dose should be subminimal (about 0.000001 mg. of purified protein deriva-

20. Wendt, H.: Klin. Monatsbl. f. Augenh. **71**:686, 1923.

21. Gilbert, W., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 42.

tive). With each negative reaction the dose is increased tenfold until a positive reaction is obtained. The resultant positive reaction is reported as +1, +2, +3, or +4, depending on the size and severity of the reaction (as suggested by Aronson), and the dose is stated. In this manner an accurate gage of the patient's degree of sensitivity is obtained. This test is called the quantitative Mantoux test (Ayman [22]) and should supplant what might be called the qualitative test. A true positive Mantoux test indicates a sensitivity to tuberculoprotein. This can only follow a tuberculous infection. It does not indicate activity or quiescence of a general lesion or the presence or lack of immunity. However, researches by Friedenwald and Dessoff [12b] indicated that patients with ocular tuberculosis exhibit a higher degree of cutaneous sensitivity to tuberculoprotein than patients with nontuberculous ocular lesions.

TREATMENT OF OCULAR TUBERCULOSIS

The treatment of ocular tuberculosis may be divided into prophylactic and curative measures.

Prophylactic measures, aimed to prevent the occurrence of ocular tuberculosis, necessarily fall in line with the widespread campaign to eradicate general tuberculosis. These measures may be specific or general.

The specific prevention of tuberculosis is being attempted on a wide scale through the use of Bacillus Calmette-Guérin (BCG). This is an attenuated tubercle bacillus which is given by mouth or hypodermically to induce specific immunity. There is much experimental evidence to indicate its efficacy, but its general adoption has been rather slow, probably because of tragic events associated with its early use. It has been used more extensively in Europe than in America.[23]

Smithburn, Sabin and Geiger [24] called attention to the unpleasant possibility, in using tuberculin M. A. 100,[25] of increasing the patient's

22. Ayman, D.: The Intracutaneous Quantitative Tuberculin Test, J. A. M. A. **103**:154 (July 21) 1934.

23. Clawson, B. J.: Experiments Relative to Vaccination Against Tuberculosis with the Calmette-Guérin Bacillus (BCG), Arch. Path. **20**:343 (Sept.) 1935. Scheel, O.: Rev. de la tuberc. **1**:529, 1935. Nègre, L., and Valtis, V.: Bull. Acad. de méd., Paris **113**:585, 1935.

24. Smithburn, K. C.; Sabin, F. R., and Geiger, J. T.: Am. Rev. Tuberc. **29**:562, 1934.

25. Tuberculin M. A. 100 is a product which was developed by Funk and Huntoon. Tubercle bacilli are grown on a special synthetic nonprotein medium. The only protein in the filtrate of such a culture is therefore synthesized by the tubercle bacilli and represents pure tuberculoprotein. The protein precipitated by ammonium sulfate is called M. A. 100. (Funk, E. H., and Huntoon, F. M.: Biochemical Studies of Bacterial Derivatives: Skin Reactions in Man with Human Tubercle Bacillus Protein M. A. 100; Preliminary Report, J. Immunol. **19**:237 [Aug.] 1930.)

sensitivity to tuberculoprotein without affording any immunization to tuberculosis.

Curative treatment of ocular tuberculosis, too, may be divided into general and specific measures.

The general measures are very important, though often neglected.

The importance of rest was emphasized by E. V. L. Brown,[26] who reported four cases of tuberculosis of the anterior segment complicating active pulmonary tuberculosis. The disease healed seemingly by treatment with rest alone.

Adequate diet, rest, heliotherapy and fresh air are as much indicated in the care of a patient with ocular tuberculosis as in the case of a patient with general tuberculosis, and in many cases the condition fails to improve until these measures are applied.

Many physical therapeutic measures are employed, among which are diathermy and the use of the infra-red rays, hot pads and the roentgen rays. Favorable reports have been made following their use when properly applied in selected cases. Therapy with nonspecific foreign proteins, such as milk, typhoid vaccine and other vaccines, and diphtheria antitoxin, has a place in certain cases, and its use has been recently discussed by F. H. Newton.[27]

Autohemotherapy, too, has its advocates. Good results have been reported of injections of blood into the anterior chamber in ocular tuberculosis, especially from abroad.[28]

In addition to the use of the well established and accepted drugs certain newer pharmacologic measures have been recommended in many reports.

Gold is being used as gold sodium thiosulfate or a double thiosulfate of sodium and gold, given intravenously, especially in cases in which the patient has not responded to tuberculin therapy.[29]

Neoarsphenamine [30] is being used with success, even in nonsyphilitic uveitis. It is recommended for treatment of patients whose condition has an obscure etiology and has not responded to other types of treat-

26. Brown, E. V. L.: Am. J. Ophth. **19**:668, 1936.

27. Newton, F. H.: Empirical Treatment of Uveitis, Arch. Ophth. **14**:618 (Oct.) 1935.

28. Miranda, A. Garcia: Arch. de oftal. hispano-am. **34**:349, 1934. Serr, H.: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **50**:41, 1934. Denig, Rudolf: Injection of Patient's Own Blood into Anterior Chamber for Tuberculous Iritis, Arch. Ophth. **14**:860 (Nov.) 1935. Kyrieleis, W.: Ztschr. f. Augenh. **85**:16, 1934.

29. Leoz, G.: Arch. de oftal. hispano-am. **35**:230, 1935.

30. Lucic, Hugo: Neoarsphenamine in the Treatment of Nonsyphilitic Inflammations of the Uveal Tract, Arch. Ophth. **15**:826 (May) 1936.

ment. Mercury and iodides, too, have their place, but the iodides must be used cautiously, if at all, for fear of reactivating old pulmonary lesions.

Specific treatment consists in the use of tuberculin. Many different forms have been suggested, but old tuberculin is probably the most widely used. Other preparations of tuberculin are as follows:

Denys bouillon filtrate,[31] preferred by Gay [32] and his colleagues at the Wilmer Ophthalmological Institute.

Bacillen emulsion (B. E.).

Tuberculin A. O., the Japanese tuberculoprotein.

Tuberculin M. A. 100, developed by Funk and Huntoon.

Beraneck's tuberculin.[33]

Although tuberculin is generally given hypodermically, it has been suggested that it be given by the percutaneous route by scarification with glass or emery paper.[33a]

The interval between hypodermic injections should be from four to seven days. The initial dose may safely be one tenth of the amount of tuberculin necessary to elicit a positive cutaneous reaction. Subsequent doses may be increased by 10 to 100 per cent to a maximum of from 50 to 100 mg. of old tuberculin or its equivalent. Severity and persistence of local reactions serve as a guide to the frequency and the size of the dose.

It is generally conceded that a definite focal ocular reaction is a serious menace to the integrity of an affected eye and calls for extreme caution in administering subsequent injections, which should be radically reduced in amount and increased later only with circumspection.

Some ophthalmologists, however, boldly seek a dose sufficient to elicit a slight focal reaction in the belief that this inflammatory reaction is necessary to fix the bacillus and heal the lesion.[34]

However, this view is not shared by most ophthalmologists, who seek to avoid gross focal reactions. They attribute the beneficent effect of tuberculin therapy either to subclinical microscopic focal inflamma-

31. Denys: Le boüillon filtré, Paris, Uystpruys-Louvain, 1925.

32. Gay, Leslie N.: The Treatment of Ocular Tuberculosis with Tuberculin, Arch. Ophth. 3:259 (March) 1930.

33. Beraneck's tuberculin is derived from a glycerinated bouillon made without peptone. This medium is less rich in albuminous substances than mediums containing peptone and hence less likely to cause false positive reactions. The tuberculin is a combination of the exotoxins from the filtrates and endotoxins extracted from the ground bacilli by a 1 per cent solution of orthophosphoric acid. (Sutherland, H. G.: Control and Eradication of Tuberculosis, Edinburgh, William Green & Son, 1911, pp. 105-118.)

33a. Löwenstein, A.: Ztschr. f. Augenh. 85:191, 1935.

34. Samojloff, A., and Tihomirova, A.: Ann. d'ocul. 172:993, 1935.

tory reactions or to the desensitization of the cells of the affected area so that they no longer are irritated by contact with tuberculoprotein.[12b]

For the malignant exudative forms of ocular tuberculosis it is deemed unwise by some to employ any tuberculin therapy at all, because of the danger of focal damage.[35]

Under these conditions only conservative local and general treatment should be employed.

There are, too, investigators who raise the question whether the good effect following the use of tuberculin may be nonspecific rather than due to specific desensitization or immunization.[36]

SUMMARY

Tuberculous lesions of the uveal tract vary greatly in their appearance and clinical course. The lesions of the iris may be nodular or diffuse. There may be marked inflammatory reaction with severe pain, flushing of the ciliary body and synechia, or the eye may be relatively quiet. There may be little exudate, or the whole posterior surface of the cornea and the floor of the anterior chamber may be filled with mutton fat deposits.

The choroidal lesions may likewise exhibit great variance in the aforementioned characteristics.

It is possible to group patients with tuberculous uveitis into three types: those whose lesions are chiefly nodular, those whose lesions are chiefly inflammatory and those whose lesions are mainly exudative. Lesions of the nodular type occur chiefly in the young up to 20 years of age; those of the inflammatory type, in persons from 20 to 30 years of age, and those of the exudative type, in persons from 30 to 50 years of age. The allergy increases in degree in a general way in the order mentioned and is the factor responsible for changing the typical picture of tubercle to that of a less characteristic inflammatory or exudative uveitis. Every patient with uveitis should be studied from the standpoint of tuberculosis. Healed lesions of the chest should be searched by the roentgen rays and a quantitative Mantoux test should be done with purified protein derivative to determine the degree of allergy.

35. Werdenberg: Useful and Harmful Therapeutics in Tuberculosis, Arch. Ophth. **13**:303 (Feb.) 1935.

36. Lockwood, Chester: Am. J. Ophth. **19**:156, 1936.

News and Notes

Advisory Board for Medical Specialties.—The annual meeting of the Advisory Board for Medical Specialties, which is the coordinating board of the twelve certifying boards in the various specialties, the Association of American Medical Colleges, the American Hospital Association, the Federation of State Medical Boards of the United States and the National Board of Medical Examiners, was held at Atlantic City, N. J., on June 6, 1937. The following officers and executive committee were elected:

Willard C. Rappleye, M.D., president, New York
W. P. Wherry, M.D., vice president, Omaha
Paul Titus, M.D., secretary-treasurer, Pittsburgh
W. B. Lancaster, M.D., Boston
R. C. Buerki, M.D., Madison, Wis.

Dr. Louis B. Wilson, of Rochester, Minn., the retiring president, was elected an emeritus member of the board.

Abstracts from Current Literature
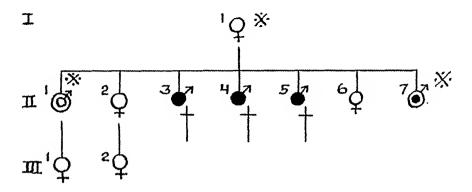
Color Sense

HEMOPHILIA AND COLOR BLINDNESS OCCURRING IN THE SAME FAMILY. W. J. B. RIDDELL, Brit. J. Ophth. 21: 113 (March) 1937.

The pedigrees of seven hemophilic families were investigated as far as practicable, and the affected members were examined for color defects. Ishihara's isochromatic plates were used in all cases, but additional tests were used for confirmation. Only one pedigree of a family

● SUBJECT TO BLEEDING
◎ COLOR BLIND
◉ SUBJECT TO BLEEDING AND COLOR BLIND
✶ EXAMINED

The pedigree of a family in which one member showed color blindness associated with hemophilia.

which showed color blindness associated with undoubted hemophilia was obtained and this was obtained from a dental clinic. The relative position of this pedigree is shown in the diagram.

W. ZENTMAYER.

Comparative Ophthalmology

THE CHEMISTRY OF THE RETINA. A. C. KRAUSE, Am. J. Ophth. 19: 555 (July) 1936.

Krause describes his experimental methods and presents a table showing the chemical constitution of the bovine retina. He comments on the various constitutents and gives the following summary:

"The chemical constitution of the bovine retina has been determined. Most of the proteins are complex lipid-proteins of nervous-tissue origin. The amount of supporting-tissue proteins is relatively small."

W. S. REESE.

MICROANATOMY OF THE EYE WITH THE SLITLAMP MICROSCOPE.
M. U. TRONCOSO and R. CASTROVIEJO, Am. J. Ophth. **19**: 583
(July) 1936.

This is the last of a series of articles. It describes the details of the
angle of the anterior chamber in primates and in man. The authors then
comment on their work and that of other investigators and give a table
of measurements found in ungulata, carnivora, monkeys and man.

W. S. REESE.

Congenital Anomalies

BILATERAL PUPILLARY ANOMALIES; PERSISTENCE OF THE ANNULAR
SINUS. V. VILLARD and H. VIALLEFONT, Bull. Soc. d'opht. de
Paris, April 1936, p. 317.

A girl of 13 years had noted a peculiar appearance of her eyes for
many years. On examination one is impressed by an anomaly of the
pupil and the iris. Externally the chestnut-colored iris was perfectly
normal, but the internal portion, which was seen best with the slit
lamp, showed a veritable border about 0.3 to 0.5 mm. wide, forming
a complete circle of blackish brown, the classic color of the uveal pig-
ment. At a distance this anomaly made the eyes appear as though the
pupillary borders were markedly irregular, the usual curved pupil
appearing serrated. Photographs and drawings of the condition are
shown. Szili and Gallenga have reported like conditions. The anomaly
is evidently caused by a developmental failure of the pigment of the
iris to cover that of the uvea.

L. L. MAYER.

Cornea and Sclera

MEGALOCORNEA. M. U. TRONCOSO and I. E. GIVNER, Am. J. Ophth.
19: 549 (July) 1936.

Troncoso and Givner define and discuss the etiologic theories of
megalocornea and report a case of this condition in a 28 year old man
who was studied thoroughly. The following summary is given:

"1. As a basis for megalocornea, disturbed endocrinology is not
supported by the accepted laboratory procedures of the day.

"2. The considerable stretching found in the ciliary body and iris
is not in accord with the theory of overdevelopment of the entire eyeball
nor even of the anterior segment in its entirety, for the size and relations
of the structures of the angle were not proportional to the enlarge-
ment of the cornea.

"3. The gonioscopic findings do not give support to the theory of
drainage obstruction, at least not by peripheral synechia. On the other
hand, the presence of hypotony in this and other cases in the literature
point to either diminished outflow or atrophy of the ciliary body due to
stretching.

"4. Cases of megalocornea presented in the future should be tabu-
lated giving specific data concerning endocrinology, X-ray, and gonio-
scopic findings."

W. S. REESE.

SCLEROKERATITIS, PROBABLY BACILLARY. F. TERRIEN and P. HALBRON, Bull. Soc. d'opht. de Paris, December 1936, p. 772.

The reason for the presentation of this patient was the good result obtained by general treatment.

A woman 25 years old had sclerokeratitis of the left eye of three weeks' duration. There existed pericorneal injection and mild infiltration of the cornea, with a small area of elevation. With local treatment it was noted that the condition extended. Four small points of scleral infiltration appeared between 3 and 4 o'clock near the limbus. General examination showed no tuberculosis, although a picture of the lungs showed perihilar shadows. The basal metabolic rate was $+ 13.5$ per cent. Successive injections of 0.1, 0.2, 0.25, 0.5, 0.75 and 1.0 cc. of methylic antigen were given. After some time visual acuity returned to normal, all vascular activity near the cornea ceased, and the slit lamp showed only slight infiltration of the corneal limbus, with absence of blood vessels. Terrien and Halborn believe that because of the course and recovery the diagnosis of tuberculous sclerokeratitis may be presumed.

L. L. MAYER.

ETIOLOGY AND TREATMENT OF KERATOCONJUNCTIVITIS SICCA. E. VON GROSZ, Klin. Monatsbl. f. Augenh. 97: 472 (Oct.) 1936.

Sjögren's syndrome is characterized by the following symptoms: dry keratoconjunctivitis, xerostomia and dry pharyngolaryngitis. These symptoms are caused by atrophy, or deficiency of secretion, respectively, of the glands. The disease occurs chiefly in women undergoing the menopause or suffering from arthritis. The findings in the cases observed by von Grosz showed that the source is not an infection but is a disease of the blood-forming organs. Characteristic signs of pernicious anemia were found in two typical cases, which suggests a connection between this type of anemia and the syndrome. Careful examination of the blood is essential in these cases. The menopause and arthritis are both caused by dysfunction of the ovaries; the condition is brought on by a disturbance of the vitamin metabolism, in the author's opinion. Study of a larger series of cases may substantiate this view. Aside from the ophthalmologic interest, the syndrome furnishes equally interesting problems for the internist, on account of the pernicious anemia; for the stomatologist, because of the xerostomia, and for the rhinologist, on account of the ozena. Therapeutic possibilities are the use of estrogenic substance, injections of acetylcholine and antianemic treatment with liver, iron and arsenic. Other therapeutic agents are vitamins A, B and C. For local application are mentioned white of the egg, fibrolysin and oil containing vitamin A.

K. L. STOLL.

Experimental Pathology

RESPONSE OF THE PUPILLODILATOR AND PUPILLOCONSTRICTOR NERVES TO MYDRIATICS AND MIOTICS IN EXPERIMENTAL HYPOCALCEMIA AND HYPERCALCEMIA. F. CASINI, Arch. di ottal. 43: 231 (Dec.) 1936.

The literature concerning the effects of calcium on the vegetative nervous system is reviewed. Hypocalcemia was induced in rabbits by

intravenous injections of a 1 per cent solution of sodium oxalate and a 20 per cent solution of sodium chloride. Solutions of pilocarpine, epinephrine hydrochloride and cocaine which were effective in normal animals were ineffective in animals with hypocalcemia produced in this way. Miosis and mydriasis could still be produced, but only with more concentrated solutions. Hypercalcemia was produced in other rabbits by intravenous injections of calcium gluconate, the excess of calcium of the blood being from 1 to 2 mg. per hundred cubic centimeters. In these animals the pupillary response to drugs remained normal. Casini concludes that lack of calcium has a depressing effect on the endings of both the dilator and the constrictor nerves. The effect is the same, as was shown with respect to other nerves, whether the nerves are sympathetic or parasympathetic. The author believes this effect takes place by means of changes in the electrochemical equilibrium of the aqueous.

<div align="right">S. R. Gifford.</div>

Experiments on Nonperforating Cauterization of the Sclera. A. de Sanctis, Arch. di ottal. 43: 252 (Dec.) 1936.

The sclera in rabbits was treated with the actual cautery 13 mm. back of the limbus for one-half minute. The tip of the cautery was 1.5 mm. in diameter and was kept violaceous to avoid too rapid an effect. The eyes were examined ophthalmoscopically and histologically, being removed at periods varying from just after the operation to ten days later. The observations were similar to those reported by other investigators after coagulation by diathermy.

<div align="right">S. R. Gifford.</div>

General

The Therapy and Pathology of Purulent Ocular Diseases. N. Blatt, Orvosi szemle 8: 411, 1935.

In hardly any part of the organism may purulent diseases bring about such serious pathologic changes and loss of function as in the eye. Such changes, unless therapeutic measures are carried out thoroughly and in due time, may lead to total loss of the eyeball. The prognosis in many cases of ulcus serpens corneae, orbital phlegmon and purulent iridocyclitis is unfavorable. It is desirable to eliminate from the nomenclature the designation "idiopathic," since many times the primary and ignored cause of the iridocyclitis called idiopathic is inflammation of the gallbladder, influenza, a dental disease or suppuration of the tonsils.

Focal infections are generally admitted to be causative of purulent changes in the eyes. However, one should not be too eager to attribute the cause to focal infections but should practice rigorous criticism. It is necessary to consider the accessory nasal cavities and to have a rhinologic examination. One should, however, not exaggerate. If the primary cause is in one of the accessory nasal cavities one must not only obtain drainage of the cavity, but pay full attention to the eye and support the entire organism against the purulent infection.

Purulent foci, though located in the remotest parts of an organism, may produce metastases in the eyes. One must emphasize, in particular,

purulent diseases of the prostate. In the absence of tuberculosis, syphilis, diabetes, leukemia, acute fever, injuries, dental disease, tonsillar infections and gastro-intestinal diseases, the technical term "idiopathic" is sometimes applied to keratitis, iritis and choroiditis when possibly the matter is a prostatic infection. One should never forget the causal rôle that disease of the prostate may play in the causation of purulent iridocyclitis.

Puerperal fever may also be causative of serious purulent iridocyclitis, and the latter may in turn be complicated, even fatally, by orbital phlegmon, thrombosis of a sinus and cerebral abscess.

Exogenous suppuration arising from an injury of the eye is likewise known as a possible cause of central fatal complications. Failure of all means of treatment has often been recorded. No success has been obtained in treatment with serums, but protective smallpox inoculation combined with subsequent injections of milk and a combination of turpentine, quinine, ethyl aminobenzoate and 84 per cent oil has proved beneficial.

Purulent iridocyclitis occurring from failure of an operation on an eye may offer the same picture as iridocyclitis due to an injury. Adequate treatment provides reopening of the wound of the operation, removal of the hypopyon, cauterization of the raw surfaces, subjunctival injection of a solution of mercury oxycyanide, treatment with serums against streptococci and pneumococci, use of ethylhydrocupreine hydrochloride as an eye lotion and intravenous injection of a colloidal suspension of silver, a preparation containing caffeine, sodium salicylate and methenamine, or the preparation of turpentine and quinine just mentioned.

As a matter of fact all the aforementioned purulent ocular diseases can heal (avoiding relapses) only if the primary focus (teeth, tonsils, prostate) is healed; the healing process is to be produced in concomitance with local treatment of the eye and general treatment. Injections of the turpentine and quinine preparation offer good results.

In seventy cases of suppuration of the eyes (purulent iridocyclitis, corneal abscess, purulent peridacryocystitis, orbital phlegmon and purulent infiltrations of the eyelids) the turpentine and quinine preparation was applied with satisfactory therapeutic results. Intragluteal injection of 1 cc. of this preparation every other day or every third day was not attended by harmful effects. Simultaneous repeated examinations of the urine are advisable, controlling possible irritation of the kidneys.

N. Blatt.

General Diseases

Ocular Manifestations in Trichinosis. E. Triandaf and J. Nitzulescu, Arch. d'opht. **53**: 47 (Jan.) 1937.

This report deals with the observation of three patients with trichinosis in one family. The symptoms were the same in all but varied in severity, the condition being acute and of long duration in one and mild and fleeting in the other two. The most characteristic symptom, which was present in all three cases, was edema of the lids and face, with an eosinophil count of from 17 to 18 per cent.

S. B. Marlow.

RUBEOSIS IRIDIS DIABETICA. O. KURZ, Arch. f. Augenh. **110**: 284, 1937.

In 1928 Salus reported on proliferation of the blood vessels in the iris of diabetic patients, which, because of its characteristics and constant association with secondary glaucoma, he classified as a definite symptom complex. Kurz reports two cases of such a condition. In one case one of the eyes had to be enucleated because of absolute glaucoma, and the author gives a description of the histopathologic changes in this eye. From his observations he has come to believe that rubeosis iridis consists in true proliferation of the blood vessels of both the pupillary margin and the ciliary region of the iris. The vessels arise in the stroma of the iris itself and not in any deposition of connective tissue. The proliferation of the blood vessels in the ciliary region leads to the formation of anterior synechiae, which are the cause of the secondary glaucoma. Diabetic retinitis is frequently accompanied with hemorrhages. These changes are most frequently seen in diabetic patients with arterial hypertension. The anatomic changes in the walls of the vessel are rather inconstant and are not specific. It is certain that thromboses, with the formation of a collateral circulation, are not the cause of this condition. Kurz believes that the formation of these vessels is an expression on the part of the tissues of an attempt to improve their damaged nutrition.

F. H. ADLER.

General Pathology

HISTOPATHOLOGIC STUDY OF CHANGES IN THE OPTIC NERVE IN PARESIS. A. BIFFIS, Ann. di ottal. e clin. ocul. **65**: 1 (Jan.); 105 (Feb.) 1937.

The literature on the subject is carefully reviewed. Biffis examined autopsy material in twelve cases of paresis. The whole course of each optic nerve, the chiasm and adjacent portions of the frontal lobe were prepared for the usual histologic stains and for the various methods of gold and silver impregnation and special stains for the nervous elements. The methods which were found to be of most value are described. Fixation was aided by injection of a 10 per cent solution of formaldehyde U. S. P. into the orbit and vitreous chamber immediately after death. The findings of ophthalmoscopic examination were available in only a few cases. Signs of atrophy of the optic nerve were noted histologically in only two cases. In one of these peripheral degeneration of fibers was present and in the other a focus of central degeneration, affecting in each case the intracanalicular portion of the nerve. In the former cases an increase in oligodendroglial elements was present, especially in the tracts of fibers affected by atrophy. In the second case an increase of these elements was present along the whole course of the nerve, being less marked about the area of central degeneration. In the other ten cases the glia, microglia and oligodendroglia showed no distinctive changes.

In this respect paresis is seen to present a marked difference from tabes dorsalis, in which atrophy of the optic nerve is a common and early finding. In paresis the degenerative changes observed bore no

direct relationship to changes in the nerve sheaths. In the diffuse degeneration seen in one case, with involvement of microglia and oligodendroglia, the primary factor seems to have been sclerosis of capillaries supplying the septums.

S. R. GIFFORD.

Glaucoma

ACTION OF CARBAMINOYLCHOLINE ON THE NORMAL EYE AND THE GLAUCOMATOUS EYE. G. DE SANCTIS, Ann. di ottal. e clin. ocul. 65: 25 (Jan.) 1937.

De Sanctis reviews the literature and reports his observations on the use of carbaminoylcholine. In normal eyes miosis was prompt, the diameter of the pupil reaching 1.5 mm. in an hour after instillation. This contraction was maintained for three or four hours, the pupil returning to normal in twelve hours. The ocular tension was reduced in normal eyes by 2 to 4 mm. of mercury, returning to normal within six to ten hours. One instillation was employed for the observations reported. Apparently the 0.75 per cent solution recommended by most authors was employed, although this is not stated. In two cases of absolute glaucoma no effect was noted on the tension, although in one case the pupil became slightly small. In one case of glaucoma secondary to hypermature cataract the tension was reduced only slightly by the drug but not to normal. In three cases of chronic simple glaucoma the effect on the pupil and on the tension was marked. The pupil contracted to 1.5 mm. in diameter within an hour, and the tension was reduced to normal within this time. The reduction of the tension following one instillation persisted for thirty to forty hours. The drug was effective in one case in which the effects of pilocarpine had been insufficient. Moderate hyperemia, with some pain in the eye and headache, was noted after the use of the drug.

S. R. GIFFORD.

Lens

EXTRACTION OF CATARACT IN 15,000 OPERATIONS. E. DE GRÓSZ, Arch. d'opht. 53: 161 (March) 1936.

De Grósz describes the operative technic which has been developed in his clinic as the result of thirty years of observation and calls attention to the increasingly better results with the development of the new technic for producing anesthesia and the better selection of cases. He outlines the Török procedure for the intracapsular extraction of cataract. Iridectomy, he believes, is safer than attempting to obtain a round pupil. In thirty years the percentage of cases of extraction of cataract in which loss of vitreous occurred rose to 2.4. For the operations for extracapsular extraction it was 1 and for the operations for intracapsular extraction 4. Within more recent years it has been decreasing (it was 2.6 for 1923-1927 and 1 for 1928-1931). Infection has been decreasing (there was a decline from 1 per cent ten years ago to 0.4 per cent in 1932). Expulsive hemorrhage was observed twelve times in thirty years, or about once in 1,200 cases. Since the use

of retrobulbar injection it has occurred more frequently. The percentage for 14,207 cases in which there were no complications was 0.087, and for the cases in which there were complications it was 0.59. Since the use of retrobulbar injection the percentage has risen from 0.1 to 0.3. As a result de Grósz has been led to pay more attention to the vascular system than formerly.

<div style="text-align:right">S. B. MARLOW.</div>

PREPARATORY IRIDECTOMY IN CAPSULOLENTICULAR EXTRACTION OF CATARACT. L. PAUFIQUE, Bull. Soc. d'opht. de Paris, February 1936, p. 115.

Paufique believes that total extraction of the lens in its capsule is the ideal operation for cataract. He has had good results in the past four years using the procedure of Arruga. This paper is a report of thirty cases of cataract in which, contrary to the usual technic, because of anticipated complications and difficulties preliminary iridectomy was done. In this group, in two cases there was glaucoma; in eight there was severe diabetes; in six there was very high myopia; in seven there were chronic iritis and pupillary seclusion, and in four, hypertension and albuminuria, and in three cases the cataract was in an otherwise normal eye, the other eye being blind. Preliminary iridectomy is done from three weeks to a month before extraction, after a small keratome incision is made near the limbus. The results in the thirty cases have been good. The advantages of this technic are as follows:

1. There is no prolapse of vitreous. 2. Extraction is easy—there is no rupture of the capsule, and the integrity of the hyaloid membrane is maintained. 3. There is no hemorrhage into the anterior chamber. 4. Most important, there is no prolapse of the iris.

<div style="text-align:right">L. L. MAYER.</div>

CHANGES IN GLYCOLYSIS IN THE LENS DUE TO AGE. L. K. MÜLLER, Arch. f. Augenh. 110: 206, 1936.

The experiments reported here were undertaken to determine whether age influenced the course of glycolysis. It was found that the capacity of an aqueous extract of lens to reduce hexose diphosphate to triose diminished markedly with the age of the lens. It was also found that the equilibrium reaction for equation hexosediphosphoric acid $\longleftrightarrow$ triosephosporic acid became impaired with age.

<div style="text-align:right">F. H. ADLER.</div>

Methods of Examination

A TRIAL SPECTACLE FOR PRISM PRESCRIPTION. I. C. MICHAELSON, Brit. J. Ophth. 21: 232 (May) 1937.

The purpose of the trial frame described is to determine in a practical way the amount of phoria to be neutralized. The frame is light and can be borne without discomfort when worn with the usual spectacles. Each cell contains a 4 prism diopter lens. Both prisms can be simultaneously rotated by a screw so as to give a maximum of 8 prism diopters. The patient is given the spectacles for home use and is directed

how to turn the screw which rotates the prisms. He is told to increase the degree of prism about 1 D. at a time until he has reached a point where maximum comfort seems to have been obtained. This is repeated for several evenings. The surgeon then has definite evidence on the basis of which to prescribe prisms and knows not to do so should the patient have obtained no relief by their trial use. There is one illustration.

W. ZENTMAYER.

Neurology

HERPES ZOSTER OPHTHALMICUS AND OPHTHALMOPLEGIA. J. VOISIN, Bull. Soc. d'opht. de Paris, June 1936, p. 399.

Voisin reports a case of herpes zoster ophthalmicus and ophthalmoplegia in a woman 73 years of age in which the third, fifth and sixth cranial nerves were involved. The syndrome originated with herpes of the left eye accompanied by ptosis. In addition to loss of sensibility of the cornea, the eye was proptosed, and movement was limited in all directions of gaze. The pupil was in a state of mydriasis and did not react. The corneal epithelium showed irregularities, and there were a few signs of intra-ocular inflammation. Visual acuity was lowered to 1/20 with correction. General physical examination resulted in no positive findings. In two months no appreciable recovery was noted. Contributions to the literature concerning patients with a similar condition by Rebattu, Monnier, Kühn, Dechaume, Bonnet and Colrat, Valière-Valeix, and Veil and Isnel are cited. Voisin believes the infection may be carried into the cavernous sinus, causing basilar meningitis.

L. L. MAYER.

CORNEAL LESIONS AFTER REMOVAL OF THE GASSERIAN GANGLION FOR TRIGEMINAL NEURALGIA. C. DRUTTER, Deutsche Ztschr. f. Chir. 248: 55 (Dec. 9) 1936.

Drutter reports that among sixty-nine patients treated for trigeminal neuritis by the injection of alcohol into the gasserian ganglion, keratitis developed in seventeen (24.6 per cent). In nine (52.9 per cent) the lesion became permanent. Eighteen patients were subjected to twenty-seven operations for removal of the gasserian ganglion. The cornea remained normal in all cases. Drutter concludes that neuroparalytic keratitis develops only as a result of anesthesia of the cornea. The cause of keratitis remains unexplained in spite of many studies. Primary trigeminal neuralgia seldom involves the first branch of the nerve. Pain in the area of this branch is considered secondary by many authors. The incidence of corneal anesthesia and consequent disease of the cornea cannot be diminished through the use of the injection of alcohol into the gasserian ganglion as developed in Germany. Subtotal posterior ganglion section of the root of the trigeminal nerve performed according to the method of Frazier and Spiller does not lead to loss of sensitiveness of the cornea and to neuroparalytic keratitis. The persistence of a high mortality in Germany following the operative intervention is due to lack of experience. Treatment with the injection of alcohol makes the operative intervention even more difficult. Subtotal section is followed by a smaller percentage of

recurrences than the injection of alcohol. According to American authors, subtotal resection of the posterior ganglion is the method of choice in the treatment of trigeminal neuralgia. Drutter suggests that the method of injection·should be reserved for patients whose general condition does not justify a major operative procedure. Younger patients should be treated, as far as possible, by operative intervention so as to avoid the possibility of keratitis.

<div align="right">J. A. M. A. (W. Zentmayer).</div>

Geniculate Bodies and Optic Cortex in Atrophy of the Optic Nerve of One Enucleated Eye. N. von Horánsky, Klin. Monatsbl. f. Augenh. 97: 438 (Oct.) 1936.

A man aged 61 had lost his left eye eighteen to twenty-two years before. The optic nerves, chiasm and optic tracts were histologically examined after the patient died from cancer of the colon. A detailed description is given of the sections stained after the method of Weigert-Pal and of Nissl. The observations in this case of peripheral blindness supported fully the view of Minkowski regarding the crossed and uncrossed fibers of the optic nerve in the geniculate bodies. The calcarine cortex on each side was found intact. The examination of the calcarine cortex did not corroborate the view of Kleist, Schröder and Henschen regarding the homolateral and contralateral termination of the visual tracts. The results obtained in this case seem to indicate that the brains of one-eyed patients cannot be used to solve the latter question, at least by the present methods.

<div align="right">K. L. Stoll.</div>

Ocular Muscles

The Stereoscope in Theory and Practice, Also a New Precision Type Stereoscope. E. Krimsky, Brit. J. Ophth. 21: 161 (April) 1937.

The considerations in the paper are grouped under two main headings—theoretical and practical.

Among the author's conclusions are the following:

Testing with a fixed stereogram (with the exception of a phoria card) is an unsatisfactory method of determining the status of fusion or for orthoptic training.

Testing with a split stereogram which can be shifted to correspond to changing positions of the visual axes with variable accommodations is the only practical method for stereoscopic study.

The ordinary professional stereoscope, because of its cramped construction and lenses of fixed focal length, is considerably reduced in its adaptability to excessive convergence and divergence. The stereoscope, however, may be so modified as to be adaptable to fairly large degrees of ocular convergence by the use of widely separated lenses of selective short focal lengths and supplemented, if necessary, by suitable prisms.

The incorporation of fixed base-out prisms, as in the ordinary stereoscope, is not, as is ordinarily supposed, a means of adapting the instrument to greater convergences but is a relic of bygone days for

employing larger stereograms (a greater field of view) for purposes of parlor entertainment. The addition of selective prisms should be left to the examiner, who alone is in a position to determine the need for supplementing base-in or base-out prisms.

The author's stereoscope enables the examiner to determine at a glance the amount of convergence and divergence, with selective accommodations, necessary for focusing split pictures, by incorporation of: (*a*) viewing lenses of variable separation and of known focal length, (*b*) a movable calibrated rod, (*c*) viewing boxes calibrated to record the amount of separation of split pictures and (*d*) tables to which the examiner may refer so as readily to translate these readings for vergence.

W. ZENTMAYER.

Orbit, Eyeball and Accessory Sinuses

TWO CASES OF INTERMITTENT EXOPHTHALMOS; VARICOCELE OF THE ORBIT. F. HIPPERT, Arch. d'opht. 53: 135 (Feb.) 1936.

Up to the time of this report ninety-one cases of intermittent exophthalmos have been described. Hippert reports two new cases, making a total of four which have been seen in Weill's clinic at Strasbourg. These two cases are fully described and are illustrated by photographs clearly showing the condition. The characteristic features are then briefly reviewed. The condition remains stationary for years, the most serious complication being hemorrhage into the orbit and into the brain. The prognosis for life is good, but that for vision is less favorable. Few patients have recovered spontaneously, and in only four cases has a cure been effected. The author favors sclerosing injections because of the great surgical risk. In neither of the two cases reported was treatment curative. Vestibular symptoms were present in one case, suggesting the possibility of vestibular varices.

S. B. MARLOW.

OCULOHYPOPHYSIAL SYNDROME FOLLOWING SUPPURATIVE SPHENOID SINUSITIS; ANATOMIC-PATHOLOGIC EXAMINATION. G. WORMS, Arch. d'opht. 53: 207 (March) 1936.

Worms presents in detail the history, course and results of postmortem examination in a case of sphenoid sinusitis of long standing which had produced visual disturbances and general physical changes constituting the essential elements of the adiposogenital syndrome. The history is noteworthy because of the grouping and predominance of symptoms which ordinarily would necessitate the diagnosis of tumor, although no tumor was found. The atrophy of the optic nerve was shown by histologic study to be the result of optic neuritis of sphenoid origin. A hypertrophic reaction of the hypophysis was also demonstrated to be the result of inflammation. The sphenoid sinusitis was completely overlooked, there being no symptoms pointing to it. The facts in the case studied demonstrate clearly that the meninges at the base of the brain and about the optic nerve can be involved in the reaction to an inflammation in the sinuses, perisinusitis.

S. B. MARLOW.

EXOPHTHALMOS DUE TO A DILATING PNEUMOSINUS OF THE ETHMOID. P. BONNET, Bull. Soc. d'opht. de Paris, February 1936, p. 109.

The term "pneumo-sinus dilatans" is reserved for a condition distinguished from pneumatocele in that a single cell of a sinus becomes filled with air and gradually increases in size. No mucocele accompanies this condition, and no liquid is found in the cell. In a thesis on this subject by Benjamins, presented at Lyon in 1935, ten cases were recorded. The present report concerns a boy 15 years old who presented himself to the clinic with exophthalmos of the left eye that had followed a severe blow to the left superciliary region. No epistaxis or ecchymosis occurred, but the proptosis was painful. It did not increase. The protrusion was 4 mm. anterior to the cornea of the fellow eye. Visual acuity was reduced to ⅛. The results of rhinologic examination, together with findings shown by a roentgenogram, warranted the diagnosis of ethmoid mucocele. The results of general physical examination were of no importance. However, at operation only a large ethmoid cell crowding the orbital contents was found. When opened this contained only air. Exophthalmos regressed fairly rapidly, and about two weeks later the exophthalmometer reading for each eye was 14 mm. There was no increase in visual acuity in the damaged eye.

L. L. MAYER.

Physiology

THE PRECORNEAL LIQUID LAYER. J. ROLLET, Arch. d'opht. 53: 5 (Jan); 111 (Feb.); 255 (April) 1936.

Rollet discusses in detail the superficial moist covering of the cornea from the standpoint of its physiologic and pathologic features, the etiologic variety of the syndromes of the preocular cavity and the therapeutic deductions which can be made. Among the various conditions that have some relationship to the fluid covering of the cornea are included neuroparalytic keratitis, ulcer of the cornea and conjunctivitis, the conjunctival and palpebral symptoms of sleep, severe general disease, the oculonasal catarrh of measles, ophthalmic zoster, intermittent pain following a slight corneal injury, filamentary keratitis, corneal lesions due to avitaminosis, corneal dystrophies and a number of other corneal disturbances. The author believes that not enough attention has been paid to the functions of this fluid, which is preocular, and the glands which produce it. It is composed not only of salts and water derived from the lacrimal glands but has properties of viscosity and adherence due to the content of mucin derived from the glands of the lids and conjunctiva. It plays an important part in the dioptric function of the cornea. A second function is the mechanical protection it affords. Most important of all, it is indispensable to the normal trophicity of the superficial epithelium. This trophic rôle must be considered from the point of view of its nervous mechanism, motor (lids), sensory (cornea) and secretory sympathetic. Alteration or deficiency in this fluid can result in anatomic lesions of the superficial corneal epithelium, accompanied by functional symptoms of which blepharospasm, photo-

phobia and tearing constitute a triad. Frequently a disturbance of the sympathetic nervous system must be suspected. In dealing with conditions affecting the epithelium much more attention should be paid to the quality of this fluid than to its quantity. In order not to disturb its formation and in case of failure of reestablishment of its secretion in integrity, one should be able to make up for its insufficiency indirectly (by a bandage, a contact glass or blepharorrhaphy) or to use a collurium which not only is bactericidal but is also eutrophic for the subjacent cells, covering them for the longest possible time owing to properties of adherence and viscosity, without the necessity of too frequent instillations.

S. B. MARLOW.

Refraction and Accommodation

HIGH MYOPIA WITH GOOD VISION. E. TRON, Sovet. vestnik oftal. 10: 19, 1937.

Tron examined two groups of eyes: (1) eyes with high myopia with poor vision and (2) eyes with high myopia with good vision. The refraction was determined with the eye under the influence of atropine; the radius of the corneal curvature was examined with the ophthalmometer; the depth of the anterior chamber and the thickness and the radii of the curvature of lenticular surfaces were measured with Tscherning's ophthalmophakometer. The refraction of the cornea and the lens and the length of the axis of the eyball were also measured. Tron reached the following conclusions:

1. The ophthalmophakometric measurements showed that axial myopia was equally prevalent in both groups of eyes.

2. The length of the axis is not the deciding factor in good vision; in some patients with high myopia with good vision the axis was of considerable length (from 29.65 to 31.8 mm.), while in a number of patients with poor vision the axis was not lengthened.

3. A few patients with high myopia with good vision had changes in the macular region, while there were no such changes in some patients with poor vision. Therefore the changes in the macula are not the only factor influencing visual acuity.

4. The refractive power of the eye is one of the important factors influencing visual acuity. The ophthalmophakometric measurements showed that in persons with high myopia with good vision the refractive power of the eye is considerably less than in persons with high myopia with poor vision, chiefly because of the low refractive power of the lens. This leads to increase of the size of the retinal image, i. e., to better visual acuity.

5. The most important factor is the unequal value of the cones in various eyes (illustrating Hecht's theory of the increase in number of the functioning cones with better illumination); most likely there is a variation of the cones in relation to their cross-section.

O. SITCHEVSKA.

Retina and Optic Nerve

SURGICAL TREATMENT OF SEPARATED RETINA BY THE GALVANIC METHOD. C. B. WALKER, Am. J. Ophth. 19: 558 (July) 1936.

This article does not lend itself to abstracting. Walker arrives at the following conclusions:

"1. An attempt to evaluate the possibilities of closing retinal tears by electrolytic alkali, by catholysis as described by Vogt, has confirmed his statements in practically every detail after 15 operations modified from Vogt.

"2. Every type of case (except the case illustrated by figure 6) described by Vogt has been observed, and three separated retinas, perhaps more extensively damaged than any included in the above report, are briefly recorded here.

"3. The upper limit of alkali treatment by catholysis was not proved, although it was believed that a record was set when 126 micro-needle cathode punctures of about one second terminal duration, carrying an average of .85 M.A. of current on a .006"-diameter needle penetrating 7 mm., were used with every evidence of success. Also 1/50 cc. of 3-percent KOH was injected subchoroidally at the macular region in the same eye. (Recently 168 micro-needle punctures for tears involving over half of the eye, were well tolerated, and resulted in improvement but not a cure.)

"4. However, failure occurred in a case with a traumatically torn hole in the retina, from two to three times the size cited by Vogt, extending centrally 45 degrees from the macula. But in two cases a large 70- to 75-degree dialysis below, also with a macular hole, was reattached, with excellent field and vision, both in a myopic eye and in a case of traumatism.

"5. Indications point to the fact that perforation of the retina is not necessary in all cases, although it is often unavoidable by micro-needle galvanic alkali treatment (catholysis).

"6. An extremely fine (.003") micro-needle supported, or trocar type of 25-percent iridium-platinum, has important characteristics as regards inhibiting leakage and bleeding.

"7. Detachable micro-needles may be used to treat more posterior areas, but they are especially valuable to relocalize in case of deceptive tracer bubbles and scleral coördination."

                                                                    W. S. REESE.

MASSIVE RETINAL FIBROSIS IN CHILDREN. A. B. REESE, Am. J. Ophth. 19: 576 (July) 1936.

Reese encountered in young persons several conditions characterized by the protrusion of a grayish white mass from the retina due to organization of hemorrhage occurring at birth or resulting from trauma after birth. He reports three cases of this condition, giving the clinical and pathologic findings, and states the following conclusion:

"Retinal hemorrhage in children can organize into a fibrous mass resembling clinically a retinoblastoma. Due to contracture, more and more of the retina can be pulled into the lesion, giving it the appearance

of progressing, and thereby leading all the more to the suspicion of a retinoblastoma. There is usually no detachment of the retina because the lesion is adherent to the choroid. The retinal hemorrhage occurs at birth from the same conditions that produce the analogous condition of massive intracranial hemorrhage. It may occur as the result of trauma in early childhood."

W. S. REESE.

CYSTS AND EPENDYMOMAS OF THE RETINA. C. DEJEAN, Arch. d'opht. 53: 81 (Feb.) 1936.

Dejean believes that the application of the principles of the general pathology of the central nervous system if applied to retinal pathology can go a long way toward clarifying disputed and obscure points. He refers briefly to the work he has already done over a period of years and calls special attention to the researches of Redslob, with whom he is in general agreement. In this report he first discusses cystic malformations of the retina, calling attention to their embryologic relations with ependyma. Cysts of the iris are included, as they are formed between the two pigmented posterior epithelial layers of the iris. He suggests that detachment of the retina can be looked on as a retinal cyst and is a reconstitution of the intra-ocular ependymal cavity by the collection of fluid from various causes. Tubular formations of the ciliary body are similarly discussed, and the conclusion is drawn that they are regressive metaplasias and that true adenoma of the ciliary body does not occur for the reason that this part does not have a secretory function. In a section on tumors of the infantile retina the author expresses the opinion that the term glioma should be rejected. If the cells resemble those of the primary epithelium of the ependymal cavity the tumor should be considered a neuro-epithelioma; if they are like those of the neurospongioma surrounding the canal the tumor should be considered a neurospongioma, and if they are like the cells already differentiated into rentinal neurons, the tumor should be considered a retinocytoma. Examples, illustrated by photomicrographs, are described. In the last section tumors of the adult retina are considered. These include pigmented epithelioma of the iris and primary ciliary and cilioretinal epithelioma. Many of these conditions can be explained on the basis of the relations of normal and pathologic homologies between the retina and the ependymal walls, or the cerebral ventricles.

S. B. MARLOW.

PRELIMINARY NOTE ON THE INNERVATION OF THE OPTIC NERVE IN MAN. J. MAWAS, Bull. Soc. d'opht. de Paris, February 1936, p. 87.

In the course of research on the vegetative nervous system Mawas made some observations concerning the optic nerve which have not previously been described. A rich nerve plexus was found surrounding the optic nerve. The name nervi nervorum opticorum is proposed for this group of fibers. An outline of the special technic necessary for the histologic study of these nerve elements is given. The author is unable to say at the present time what the exact destination of the fibers is, but it is probable that they subserve the membranes and vessels in

the vicinity. Two excellent photomicrographs accompany the article. Mawas believes that further study by him will answer the question of the function and destination of these nervi nervorum opticorum.

L. L. MAYER.

THE REFLEX STREAKS ON THE RETINAL VESSELS. ALFRED JAEGER, Arch. f. Augenh. 110: 137, 1936.

Being a physiologist and not an ophthalmologist, Jaeger has studied the light streaks on the blood vessels in the eye by comparing them with similar streaks on the mesenteric vessels of lower animals. His chief interest has been to show that the moving column of blood in the vessels is one of the chief factors in producing the bright light streak in the arteries. He believes that the central portions of the column of blood which are moving more swiftly tend to orient the blood cells in such a way that they all lie with their broad surfaces in the long axis of the vessel, and this makes for a more perfect reflecting surface than the cells which line the walls of the vessels. These cells, moving slowly, are apt to have their edges turned toward the observer, or irregularly placed so that they do not reflect the light as well.

Jaeger concludes that the general background of the vessels is important and that the walls of the vessels, although translucent, cast a shadow which sets off the central light streak. In the frog, for example, which shows a relatively dark background, the central light streak is not nearly as bright as the two reflections from the walls of the vessels. In man and in the dog there are two kinds of reflex streaks: 1. A small bright bluish white streak which comes from the membrana limitans interna of the anterior wall of the vessel. This is often broken corresponding to the curvature of the vessel and is found in man on the veins as well as on the arteries. 2. A broad yellowish red streak which is reflected through the wall of the vessel from the retina and which is reflected back to the observer from the column of blood because of its properties, which have already been mentioned. It is found only in the arteries in man and is absent in these when the column of blood has stopped moving.

These two light streaks can be differentiated from each other by moving the light of the ophthalmoscope on the vessel under observation. The first one follows the movement of the source of light clearly, while the latter remains stationary.

F. H. ADLER.

ORIGIN AND SIGNIFICANCE OF GUNN'S SIGN. E. HORNIKER, Klin. Monatsbl. f. Augenh. 97: 315 (Sept.) 1936.

Horniker reviews the publications on Gunn's sign, beginning with this author's paper. A complete bibliography on the subject and on related questions is appended. Referring to the research of Salus and to that done jointly by Sallmann and Kahler, Horniker describes in detail modifications in the use of palpatory sphygmomanometry by means of Boulitte's apparatus and the manner in which he employs oscillometry after the method of Plesch. After describing these methods

and other oscillatory methods adduced for comparison, Horniker states his own views on the vascular condition in patients who present Gunn's sign and Salus' arc. He points out that pulsatory phenomena in the retinal vessels may be observed even in healthy persons not only with the large ophthalmoscopes but also with the small electric ophthalmoscope. Jonas Friedenwald's terminology of the pulsations is discussed and commented on favorably.

With reference to the origin of Gunn's sign, Horniker distinguishes two phases. Atony of the central retinal artery, which possesses only a few muscle fibers, precedes arteriosclerosis and is of great importance during the first phase. The first pathologic changes are produced by increased expansive pulsation in the wall of the artery.

The second phase is the hypertony that occurs with advanced age; it narrows the bore of the artery by increased tonus. Then the walls of the veins, which were compressed by preceding changes, become visible at a certain distance from, and on both sides of, the artery, thus producing Salus' arc. Hence the initial stage of arteriosclerosis plays a predominant rôle in the genesis of Gunn's sign. Discussing the changing location of the retinal vessels which takes place during the course of observation, as described by Westkamp, Horniker suggests that photographs be taken systematically for several years. Simultaneously, oscillograms and ophthalmodynamometric data should be recorded.

<div align="right">K. L. STOLL.</div>

Trachoma

TRACHOMA. R. E. WRIGHT, Brit. J. Ophth. 21: 198 (April) 1937.

Wright briefly reviews the results obtained from experiments based on the observation of the appearance of growths on the chorio-allantoic membrane of the chick when inoculated with unfiltered trachoma material. The laboratory aspect of the work was side-tracked since controls revealed that a variety of agents other than known viruses and unfiltered trachoma material produced similar growths on the chorio-allantoic membrane with varying histologic changes. It may be that these findings put an end to the original idea, but until the whole question of the effects of different agents (organismal, chemical, mechanical known virus, etc.) on the chorio-allantoic membrane is worked out, nothing further can be said as to the significance of the effect of inoculation with unfiltered, but apparently bacteriologically sterile, trachoma material.

<div align="right">W. ZENTMAYER.</div>

TRACHOMA AS A VIRUS DISEASE. A. F. MACCALLAN, Rev. internat. du trachome 14: 5, 1937.

Inclusion bodies, noted in cases of trachoma in 1907, have been since found in all virus diseases, but only in such diseases. Cellular inclusions have two components: (1) gram-negative elementary bodies, Chlamydozoa, consisting of dustlike granules from 200 to 300 millimicrons in diameter, embedded in a ground substance capping the nucleus and (2) larger initial bodies, about the size of a minute coccus, which may or may not be free. Both bodies are considered different forms of the

same organism. The prevention of trachoma lies in the avoidance of contagion, and the disease is probably contagious until the late phase of stage III is reached.

J. E. LEBENSOHN.

PATHOLOGIC CHANGES IN LATE TRACHOMA. A. BIRCH-HIRSCHFELD, Rev. internat. du trachome 14: 13, 1937.

Characteristic of advanced trachoma is an excessive neoformation of fibroblasts occurring first in the subepithelial and pretarsal tissue and later involving the tarsus. Since the proliferation of fibroblasts is the sequel of the process of infiltration by lymphocytes and plasma cells, the serious sequelae of trachoma are best avoided by early energetic treatment.

J. E. LEBENSOHN.

TRACHOMA AND BACTERIUM GRANULOSIS OF NOGUCHI: COMPLEMENT FIXATION TEST IN TRACHOMA. E. TRAPEZONTZEVA, Sovet. vestnik oftal. 10:101, 1937.

The complement fixation test was done with the immune serum of rabbits and with serums of patients suffering from trachoma. The culture of Noguchi's bacillius was used as an antigen; its antigenic and agglutinable character was studied previously in view of its anti-complementary properties. The antigen was prepared in various ways: by irradiation with ultraviolet rays, by heating and by treatment with solution of disodium sulfate. It was carefully titrated. The serum of healthy persons served as a control.

Two hundred trachomatous patients were examined. The complement fixation test was positive in 57 per cent. These included patients with progressive trachoma and those incompletely cured. The complement fixation test was negative in doubtful cases or in those cases in which cure was temporary or permanent. The reaction of the control serum was also negative. The reaction was positive in the following percentages in cases classified according to the various stages of trachoma: trachoma of stage 1, 34.5 per cent; trachoma of stage 2, 75 per cent, and trachoma of stage 3, 51.8 per cent. In order to obtain accurate data the serum had to be fresh and inactivated not longer than one or two days previous to the test. The reaction should be read as soon as hemolysis is discovered in the test tubes containing the control.

O. SITCHEVSKA.

Tumors

LYMPHOSARCOMA OF EYELID. V. M. MÉTIVIER, Brit. J. Ophth. 21: 202 (April) 1937.

Sarcoma of the eyelid is a rare growth, but Smith and Elmes in an analysis of five hundred tumors among the natives of Nigeria found ten cases of round cell sarcoma of the orbit. These authors suggested that probably many of these growths had their origin in trauma, since injuries to the eyes are notably common among natives.

Métivier reports a case of lymphosarcoma of the eyelid. The patient was a brown-skinned boy of impure African descent living in Trinidad.

When he was first seen the condition appeared to be a large untreated chalazion. Antisyphilitic treatment was given, as the Wassermann reaction was strongly positive. No effect was noted from the treatment, and as the growth at that time completely hid the globe and there was an extension toward the subconjunctival tissue of the lower lid, with invasion of the caruncle and plica, exenteration of the orbit, with removal of the eyelids, was done. Histologic examination proved the growth to be a large round cell sarcoma. An enlarged lymph node on the angle of the jaw showed the same structure. Two years later there was no evidence of recurrence and the patient was in good health.

This article is illustrated.

W. Zentmayer.

Sebaceous Tumors of the Lids. G. Morard, Bull. Soc. d'opht. de Paris, June 1936, p. 435.

Morard reviews the literature on tumors developing in the sebaceous glands of the lids. The embryologic aspects of these glands as discussed and pictured by Ida Mann is commented on. On the basis of an anatomic study of the meibomian and ciliosebaceous glands, the cellular elements are divided into four groups—generative cells, transitional cells, sebaceous cells and reticular formation.

Morard groups the cutaneous sebaceous tumors into sebaceous adenoma (typical and metatypical) and sebaceous epithelioma (basosebaceous, spinosebaceous and mixed metatypical and sebaceous).

The meibomian tumors are classified into adenoma and epithelioma.

Reports of cases are included, illustrated with excellent photographs showing the clinical and microscopic features. The conclusions are that the embryologic and histologic analogies between the meibomian glands and the cutaneous sebaceous glands establish a parallel between the neoplasms developing along these two orders of formation. Therapeutically, the sebaceous meibomian epitheliomas are markedly radiosensitive as compared with the cutaneous sebaceous epitheliomas. An excellent bibliography is appended.

L. L. Mayer.

Therapeutics

Effect on the Eye of Radium Used for Treatment of Malignant Disease in the Neighborhood. Philippa Martin, Brit. M. J. 1: 651 (March 27) 1937.

Martin has studied the effect of radium on the eye in cases of tumor of the skin and of the upper jaw in which this treatment was used. The treatment consisted in part of interstitial irradiation with needles of low linear intensity or with a 1 Gm. radium unit. Needles should have a screen of 0.8 mm. of platinum in all cases in which it is necessary to introduce radium into the orbit. The reactions appeared in the conjunctiva either early or late; congestion of the iris occurred, which was never serious, and necrosis of the cornea was seen, which was the most serious result and was usually associated with radium necrosis of skin or bone. The necrosis of the cornea bears some resemblance to neuroparalytic keratitis; it rarely takes place rapidly. If the cornea becomes affected it should be protected by a suture of the eyelids.

Radium cataract is usually a late reaction, occurring two or more years after exposure. Martin mentions an unusual case of early mature radium cataract. To protect the eye when the surrounding tissues are to be irradiated it is best to stitch the lids together. The lids should be split and the sutures left in place for four weeks. The use of the 1 Gm. unit produces so little reaction that preventive treatment is not necessary. It is well to keep the eye under observation in a case in which it has been subjected to intense irradiation, as necrosis of the cornea generally comes on late, namely, about eight weeks after exposure.

The author concludes as follows:

"The observation of eyes which have been exposed to radium irradiation of destructive intensity has demonstrated a series of morbid changes leading progressively to radium necrosis of the cornea and loss of the eye

"When treatment of neighbouring malignant tumors is by interstitial irradiation these changes can be reduced by adequate screenage of the radium needles.

"After mass irradiation by a large quantity of radium at a distance the damage to the eye is negligible.

"As greater quantities of radium become available, and facilities for the use of mass irradiation become more widely distributed, it is to be hoped that damage to the eye by interstitial irradiation will cease to be of more than historic interest."

A. KNAPP.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

Forty-First Annual Meeting, New York, Sept. 26 to Oct. 3, 1936

FRANK E. BURCH, M.D., St. Paul, *President*

WILLIAM P. WHERRY, M.D., Omaha, *Secretary*

CLINICAL PROBLEM OF ALLERGY IN RELATION TO CONJUNCTIVITIS AND IRITIS. DR. ALAN WOODS, Baltimore.

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PRIMARY SARCOMA OF THE UVEAL TRACT: AN ANALYSIS OF TWENTY-SEVEN CASES. DR. S. HANFORD MCKEE, Montreal, Canada.

In the uveal tract, sarcoma appears much oftener in the choroid than in the iris or the ciliary body. E. Fuchs (quoted by Greeff: Pathological Anatomy, Berlin, A. Hirshwald, 1903, p. 296) estimated that in 6 per cent of the cases of sarcoma of the uveal tract the iris was affected; in 9 per cent, the ciliary body, and in 85 per cent, the choroid.

Malignant melanoma is relatively rare. According to Fuchs (Das Sarcom des Uvealtractus, Vienna, W. Braumüller, 1882), it occurs in about 0.066 per cent of all cases of ocular conditions. Stallard (in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 689) stated that during 1925-1931 at Moorfields sarcoma of the choroid was found in 1 of every 4,000 patients attending the clinic for outpatients. It occurs mostly between the fortieth and sixtieth years, though in rare instances it has been found in children. Males and females are affected about equally. Rarely does this disease affect both eyes.

The origin of the essential pigment of the melanin of the eye may have an important pathologic bearing on the nature of intra-ocular tumors: Bloch isolated from the embryo of the broad bean 3, 4- dihydroxyphenylalanine, a substance which he called dopa, and showed that it was readily changed by this oxidase to melanin. When this substance is added to epidermal cells of skin in frozen sections fixed in formaldehyde, granules of melanin are formed (the dopa reaction).

Melanin is closely allied to epinephrine, and it is probable that the two substances are derived from the same precursors and form alternative end-products in metabolism. When the cells are stimulated to proliferate so that there is an increase of the oxidative ferment, a pigmented tumor is the result, or generalized melanomatosis may follow, as in Addison's disease. It would seem from clinical observations that in

the eye melanogenesis is a function of both epiblastic and mesoblastic tissues, and recent chemical work seems to substantiate this deduction. The chemical findings fall in line with the clinical observations, and, so far as the eye is concerned, it seems clear that pigmentation may be either ectodermal or mesodermal, so that malignant melanoma may be either carcinomatous or sarcomatous. In respect to this Terry and Johns (*Am. J. Ophth.* 18: 903, 1935) stated: "In recent years grave doubts have arisen as to the mesoblastic origin of the class of primary malignant uveal neoplasms commonly called sarcomata. In fact, Masson and others have presented strong evidence that is indicative of ectodermal origin."

In 1931 Major Callender (*Tr. Am. Acad. Ophth.,* 1931, p. 131) described four specific types into which all primary malignant uveal neoplasms can be classified:

1. *The Spindle Cell Type.*—The characteristics of this type of tumor are sheets, whorls and other irregular arrangements of spindle-shaped cells with long oval nuclei. The ends of the cell appear to terminate in fibers, so that the cells resemble fibroblasts. There is no argentophil reticulum except that accompanying the nutrient vessels. These spindle cell tumors can be separated into the following two divisions by the nuclear characteristics.

Subtype A: The nucleus has a delicate reticular structure in which the nucleolar material is not well defined. These tumors are usually fairly heavily pigmented.

Subtype B: The cell has a sharply defined, deeply stained, small round nucleolus, usually situated near the center of the nucleus in a rather coarse nuclear network. These tumors are usually lightly pigmented.

2. *The Fascicular Type.*—The cells of this type of tumor are elongated, sometimes forming fiber-like structures, but rounded and polygonal shapes are seen. The characteristic nucleus is oval, has a distinct nucleolus and closely resembles the nucleus of the cell of the spindle cell tumor of subtype B. The majority of the cells are arranged in columns or fasciculi, the long axis of the cell being at right angles to that of the column. The cells radiate about the center of the column in a palisade arrangement, the center being a lymphatic or a capillary blood vessel. Pigmentation is usually scanty.

3. *The Epithelioid Type.*—Tumors of this type have polygonal cells, usually of relatively large size, though there is considerable variation in both the size and the shape. The nucleus is large and round or somewhat oval, and the nucleolus is distinct. This type varies greatly in the degree of pigmentation.

4. *The Mixed Cell Type.*—Tumors of this type have irregular mixtures of cells of the spindle and epithelioid types, with occasional areas of cells of the fascicular type. These tumors are heavily pigmented.

A recurrent orbital tumor has been noted rarely, and in this series was present in only 1 case. Terry and Johns suggested that the orbit may be an unfertile field for tumors and that Tenon's capsule may hold the tumor in check at least long enough for a metastasis already existing or a metastasis from the recurrent orbital growth to bring about a fatal issue before local recurrence manifests itself.

The liver is the most fertile field for a metastatic growth, and while most metastatic tumor cells are blood borne, metastasis by way of the cervical glands does occur.

DISCUSSION

Dr. T. L. Terry, Boston: Primary tumors of the iris appear to be less malignant than those of the choroid, and they should be regarded as in a different category.

I wish to disagree with the essayist and the many others who insist on dividing tumors of the uveal tract into the four stages. Such a division is not only artificial but also extremely misleading. The term "stages" immediately brings to mind a picture of development and metamorphosis in a sequential order. It also tends to lend a false sense of security if the eye is enucleated when the tumor is small, the retina unseparated, glaucoma not present and the eye unruptured. In the series reported by Johns and myself in 1935, stage four (metastasis) was reached in 1 instance even before the complete development of stage one, i. e., before separation of the retina. Stage four (metastasis) was reached before stage two (glaucoma) in 7 instances. And, further, stage four (metastasis) was reached before the advent of stage three (extension of the tumor outside of the globe) in 17 instances. It must be stressed that metastasis may occur early, even before retinal separation. One is never sure of cure by enucleation. The prognosis cannot be judged by either the size or the so-called stage of the tumor. Therefore, it is never safe to delay enucleation after the diagnosis is made.

The most striking feature of Callender's classification according to cellular type is that not in a single case of tumor of the spindle cell subtype A did death occur. Is this type a benign melanoma of the choroid, capable of destroying the eye and even invading the orbit? The differentiation of the other types may eventually give a fairly accurate method of grading malignancy. A study of the reticulum as stained by the silver stain of Wilder is of value. The value of the Klein test as applied by von Hippel to malignant melanoma of the uveal tract must still be proved.

When one analyzes the paper of the essayist one finds much of practical clinical information. It shows the importance of the following points, which I stress at every opportunity.

1. One should suspect malignant melanoma in every case of separated retina.

2. One should suspect malignant melanoma in every case of unilateral glaucoma.

3. One should suspect malignant melanoma in every case of unilateral uveitis. In tumors of this type, when there is a great deal of necrosis or even a moderate amount, uveitis is likely to occur. Of course, it is unilateral.

4. One should suspect malignant melanoma in every blind eye, especially when it is impossible to see the fundus. A number of ophthalmologists advocate operating on a blind eye with glaucoma in order to reduce the pain and make the eye comfortable, but in our experience of 97 cases a tumor was found after the eye was opened in some 40 instances, and it was not even suspected clinically. I have talked

about this so much at our local institution that the house officers now record for almost every eye that is removed a note of "possible sarcoma." That may be something of a joke, but at least it is in their minds for clinical differentiation at the time.

5. Finally, it should be remembered that an intra-ocular operation may stimulate the tumor to grow more rapidly and become more malignant, as well as furnish a tract of decreased resistance through which it may grow. In fact, some observers have stated that the most common channel by which the tumor extends from the eye is along the tract of an operative wound.

Sir John Herbert Parsons, London, England: The ordinary and flat sarcomas, I think, are somewhat like the secondary carcinomas which grow in between the planes of choroid tissue. I think they are located among the lymph spaces of the choroid, and this points to the origin of the flat sarcomas, which also have a sort of alveolar structure: In all probability, the flat sarcomas, which are rare, are endotheliomas growing in the tissue spaces.

With regard to malignancy, there is, first, the question of pigmentation to be considered. I think that pigmentation has little reference to malignancy in cases of sarcoma of the choroid. Most of these tumors are pigmented, but one does see malignant true leukosarcoma. Some of the pigmented tumors are really leukomas which have hematogenous pigmentation. They are probably usually of the angioid type, not in the ordinary sense of the word but in the sense that they are of the vascular type of sarcoma. In tumors of this type the pigmentation is of a different kind. Even in such tumors the hematogenous pigmentation may become iron-free, and it would be impossible to distinguish it from the other type of pigmentation. I do not believe the pigmentation has much to do with the malignancy, but the fact that the growth is pigmented makes one extremely suspicious of malignancy, unduly so. That is due to the fact that a vast majority of tumors are pigmented.

The most important point about a malignant growth is the site of the tumor in the eye. If it is discovered when it is at an early stage, the eye can be enucleated without delay. I think, from my experience, that in these cases the prognosis is good with regard to subsequent developments. But if the tumor is situated at the periphery it is less likely to be detected as early as a central growth. Consequently there is delay, which is often considerable, before the eye is removed, and the prognosis is much worse than in the case of a central tumor.

Clinical Importance of Pigment in the Fundus. Dr. Arthur J. Bedell, Albany, N. Y.

Variations in the pigmentation of the fundus assist in the diagnosis of ocular diseases and are often a reflection of the condition of the general health. The color of the fundus depends on the amount of pigment in the hair and skin. Congenital deposits of pigment are of no pathologic significance but may confuse the uninitiated. They are flat collections of pigment situated near the disk and peripherally. Senile pigmentation is an exaggeration of drusen, Tay's choroiditis and senile macular degeneration. The benign drusen may become so large and so central as to confuse the ophthalmologist who does not use the dis-

criminating tests for vision and defects of the field. Inflammation of the retina is always accompanied by migration of the pigment. The picture depends on the number and size of the patch or patches. Many characteristic forms have been described during the eighty years of ophthalmoscopy. When the retina is detached, the appearance of unusual pigment may be the determining factor in diagnosing an underlying malignant growth. All penetrating wounds of the sclera and all severe contusions of the globe leave pigmentary evidence of damage. The degree and site of the lesion are often of great medicolegal value. Of particular importance is a complete understanding of the pigmentation in syphilis and in retinal hypertension. Retinitis pigmentosa, retinitis punctata albescens and choroideremia should be separated and not grouped as evidence of a single cause until the cause has been convincingly demonstrated and proved.

DISCUSSION

Dr. W. E. Fry, Philadelphia: There has been some difference of opinion concerning the origin of the pigment in the fundus. Some have considered that the pigment originates in the pigment epithelial cells and migrates from there into the choroid. Others have considered that the pigment originates separately in the retina and in the choroid. The latter opinion is probably correct. By means of the dopa reaction Black found that only epidermal cells gave a positive reaction; the mesodermal cells gave a negative reaction. He therefore concluded that the pigment in the choroid results from migration from the retina. However, embryologically it has been shown that the pigment appears very early in the retina, i. e., at the fifth week, and very late in the choroid, at the fifth to the seventh month after the development of the lamina vitrea, and that it appears first in the outer layers of the choroid. In the pigmented epithelium the pigment arises on the cytoplasm and is in the form of a melanoprotein. Pathologically, pigment may appear in other cells, as in fibrocytes and also in clasmatocytes of the retino-endothelial system. The pigmentation of the former cells is always finer and less dense than that of the latter. In certain conditions, such as disturbances of nutrition, the chromatophores, which normally have branched processes, lose their processes and have the appearance of clump cells. This has led various authors to believe that these two cells are variations of the same cell. The pigment in the eye is not an inert substance but has certain irritative qualities, and immunologically it is capable of acting as an antigen. The former ability is shown when pigment is injected intra-ocularly, when it produces a certain amount of retinal degeneration. The latter quality has been seen in relation to sympathetic ophthalmia.

Certain analogies have been drawn between dermatologic and ophthalmic conditions. The retinal pigment epithelium has been compared to the epidermis, and the uveal tract to the corium. A particularly important relationship has been shown between angioid streaks of the retina and pseudoxanthoma elasticum. Each of these conditions is rare in its field, and their occurrence together shows a relationship of great importance. There have been certain facts brought out concerning pigmentation in the skin and vitamin C and copper. There are cutaneous conditions, namely, scurvy and Addison's disease, that have as one

feature increased pigmentation. It has been found that the administration of vitamin C will prevent the hyperpigmentation shown in these diseases. An idea of the site of the reaction is obtained by the use of dopa. Vitamin C interferes with this reaction. In the formation of pigment, copper apparently acts as a catalyst, and it has been shown that a definite proportion exists between the amounts of pigment, vitamin C and copper present in the skin. In view of the fact that when pigment is introduced into the eye it causes retinal degeneration, vitamin C therapy may be of use in the treatment of conditions characterized by retinal hyperpigmentation, in spite of the fact that the pigment may be only a secondary factor.

DEFECTIVE CENTRAL VISION AFTER A SUCCESSFUL OPERATION FOR DETACHMENT OF THE RETINA. DR. ALGERNON B. REESE, New York.

It is common experience that after a successful operation for detachment of the retina the central vision is frequently found to be defective. The statistics of Dunnington and Macnie, compiled from cases of retinal detachment in which operation was performed, observed at the Institute of Ophthalmology, New York, were reviewed with the idea of determining what percentage of the patients in whom reattachment occurred regained normal central vision and whether or not the duration of the detachment affected the central vision after reattachment. Of the 55 patients in whom reattachment occurred, 18, or 32 per cent, obtained central vision of 20/30 or better. There were 32 patients in whose cases the duration of the detachment had been one month or less, and of these, 12, or 38 per cent, obtained central vision of 20/30 or better. There were 23 patients in whose cases the duration of the detachment had been over one month, and of these, 6, or 26 per cent, obtained central vision of 20/30 or better.

From these figures one can deduce that approximately normal vision (20/30) is obtained in only about one third of the cases in which operation for detachment of the retina is successful and that the duration of the detachment is a factor in the visual result.

From microscopic examination in the routine cases of retinal detachment in which the eye is enucleated, it is well known that cystic spaces occur in the macular region and, to a less extent, elsewhere in the retina. This change is usually considered a late sequela, and its occurrence has been noted almost invariably in eyes in which there was some other pathologic change, so that it is difficult to say to what extent detachment alone was responsible for the cysts and to what extent other factors were the cause.

Twelve patients with simple serous detachment of the retina ranging in duration from less than two weeks to four years all showed cysts in the macula. It is here that the cysts seem to appear first and to attain their largest size. In the cases in which the detachment is of longer standing—of three months' duration or longer—they are frequently also found elsewhere in the retina. They are situated primarily in the external plexiform layer, but they are often seen to a lesser degree in the internal nuclear layer. At first the retinal elements seem to be separated merely by edema, which localizes into small cystic spaces.

These coalesce so that ultimately the cysts become larger and separated from contiguous cysts by thin septums of glial tissue. The content of the cysts is usually a homogeneous, colorless fluid which stains weakly with eosin in some instances, particularly when the condition is of long standing. This, no doubt, represents an increase in the albuminous content due to inspissation.

Cysts in the macula or elsewhere in the retina cannot be recognized clinically as such by the ordinary ophthalmoscopic examination. They may appear as poorly demarcated foci ranging in color from grayish to yellowish white or as a rarefied area described usually as a "hole." Vogt has recognized them as cystic spaces by means of red-free light and has described their appearance in cases of retinitis pigmentosa, thrombosis of the central vein and detachment of the retina.

In most instances, what is diagnosed clinically as a hole in the macula represents conglomerate or confluent cysts.[1] This statement is based on the following facts:

1. One often sees in the center of a so-called hole in the macula a foveal reflex, which apparently comes from the anterior limiting layer of the cyst.

2. By means of red-free light, or by spectral illumination with the ophthalmoscope, one can usually see cystic spaces surrounding the so-called hole, and sometimes it can be detected that the irregular borders of the hole are continuous with the surrounding cysts. Occasionally, septums can be seen in the hole.

3. Actual holes in the macula almost never are seen in microscopic sections, whereas cystic spaces are common.

The following conclusions may be stated:

1. Cystic degeneration of the macula accounts for the poor central vision after the reattachment of a detached retina which involved the macular region.

2. The defective central vision frequently found after a successful operation for detachment of the retina suggests that macular detachment with formation of cysts was present, even though it might not have been recognized clinically.

Cysts or holes may also appear in the macula as follows: 1. Following trauma. In this condition the cysts are thought by most observers to be due to localized edema, which leads to pressure atrophy of the affected retinal element, with the formation of small cystic spaces, the walls of which form larger ones by confluence. Sometimes the cystic spaces are filled with hemorrhage. A hole may be present after commotio retinae. 2. As a complication of iridocyclitis. In this condition toxins apparently reach the macular region and give rise to inflammatory edema. These cysts may be transitory and may reappear. 3. Accompanying retinitis pigmentosa. In this condition there may be cystic formations in the macula, usually in the early stage of the disease. These may be transitory and are usually attributed to edema. 4. Following occlusion of the central vein of the retina. Cysts in the macula are extremely common in this disease. They are due to edema, and sometimes they are filled with blood. 5. As a complication of retinal arteriosclerosis. This condition occurs in older persons, and the cysts are attributed to edema due to arteriosclerosis. They may appear in the early stage of senile macular degeneration. Arteriosclerosis due to syphilis may also be the cause of macular cysts.

DISCUSSION

DR. CLIFFORD B. WALKER, Los Angeles: The cystic degeneration that occurs in Henle's layer, that is, the plexiform or internuclear layer, was first noted by Bruno in 1902 in connection with melanosarcoma of the choroid. It was repeatedly noticed until 1910, and in 1916 Vogt noticed the cystic degeneration taking place at the macula in the case of carcinoma, and he wished to connect it with the separation of the retina, with the thought of possibly showing the mechanism of the detachment.

One can see that the layer of Henle, which is marked at the macula and constitutes most of the tissue there, because the two layers have been pushed aside, has degenerated in such a way that there are a top layer of limiting membrane and a bottom layer, and there are intermediate stages, as can well be imagined, which might affect the external limiting membrane in such a way that three layers may occur—laminated holes, I shall say, for lack of a better phrase now.

I shall speak concerning the pebbling that is noticed around the hole. If this is cystic in character, it is exceedingly regular in outline. But the cysts, as is noticed, are not so regular, so uniform or so able to indicate the pebbling like Scotch grain leather.

The pebbling is due to toxic effects. When one watches the same process in capillary degeneration, there is edema, with early stages of cystoid degeneration. But in regard to the pebbling that occurs under the conditions found in the retina, it seems to me reasonable to regard it largely as a pattern of the basework of the retina and as an underlying edematous and pyoid degeneration rather than as cysts in that particular spot and with that particular arrangement.

DR. HARRY S. GRADLE, Chicago: The lack of central visual acuity after a successful operation for detachment of the retina may be due to one of three things. In the first place, there may have been a preoperative disease involving the macula. This possibly might have been seen beforehand, but if the detachment was sufficiently extensive to elevate the macula it would have been impossible to observe it.

Second, a postoperative change in the macula is usually subsequent to hemorrhage. Hemorrhage into the macula following operation for detachment of the retina is not uncommon.

Third, patients who on ordinary ophthalmoscopic examination show a normal macula may have a lack of central visual acuity and a definite central scotoma often from 3 to 5 degrees in size.

Since April I have observed 4 cases of defective central vision following a successful operation for detachment of the retina in which I have been able to detect the cause of the failure of macular vision. I have not been able to see these cysts with the ordinary ophthalmoscope but have seen them with the Friedenwald slit ophthalmoscope when used, as Dr. Reese has described, with retro-illumination (if one wishes to call it that) and particularly when used with the red filter—and I said red, not red-free. The details of such cysts do not appear with the red-free filter but appear clearly when the red filter is used.

In these 4 cases the cysts lay directly in the macula and varied from one quarter to a little more than one third of a disk diameter in size. In every instance they were regular and oval, vertically oval. This was

probably a coincidence. At no time did I find the irregular walls as in the last picture shown by Dr. Reese. The cysts were elevated, and the elevation could be detected by the use of the extremely fine slit. There seemed to be a thin but definite anterior wall, which, apparently, varied in thickness in the different cases. The surrounding macula was flat, and, as far as could be detected, there was no elevation of the retina in or around the macula except at the cyst.

In this connection, may I speak of one other observation that I have made in 2 cases? On the fifth to the ninth day subsequent to detachment of the retina or to operation for detachment of the retina there has appeared massive detachment of the choroid. This persisted in 1 instance for four weeks. In another instance it persisted for three weeks, and was followed by successful reattachment of the entire retina, with good central visual acuity.

From the mechanical standpoint, it is easy to see why choroidal detachment is a desirable feature, because, whatever surgical procedure one performs for detachment of the retina is for the purpose of producing adhesion between the retina and the choroid, for the production of which adhesion the retina must either be in apposition with the choroid or close thereunder.

If a large amount of subretinal fluid is present, it is obviously impossible for the retina to be caught in the adhesive process and retained there. Consequently, there is the possibility that certain types of retinal detachment may be handled by the artificial production of detachment of the choroid, which, of course, is spontaneously limited.

A New Operation for Chronic Glaucoma. Dr. Otto Barkan, San Francisco.

This article was published in full in the November 1936 issue of the *American Journal of Ophthalmology,* page 951.

Visual Sequela Resulting from Meningococcic Meningitis. Dr. Parker Heath, Detroit.

This article will be published in full later.

Nodular Dystrophy of the Cornea. Dr. Benjamin Rones, Washington, D. C.

Groenouw (*Arch. f. Augenh.* 21: 281, 1890) first described nodular dystrophy of the cornea (*knötchenformige Hornhauttrübungen*) as consisting of numerous small, roundish, gray, nonconfluent opacities in an otherwise clear cornea. These tend to involve the central portions, leaving the periphery clear. The larger nodules cause the epithelium to bulge forward and become irregular. These opacities remain unchanged for many years, usually without inflammatory changes. Both eyes are always affected, and the disease sometimes occurs in several members of the same family.

The type of nodular dystrophy described by Salzmann (*Ztschr. f. Augenh.* 57: 92, 1925) differs from the aforementioned condition in that the nodules are larger and less numerous and are markedly elevated above the surface of the cornea. It occurs usually in women and is not

familial. In most of the reported cases the condition was unilateral, and there was a history of repeated attacks of eczematous keratoconjunctivitis. Salzmann expressed the opinion that this variety of dystrophy is not as rare as the paucity of reported cases leads one to believe.

The lattice type of corneal dystrophy was first reported by Haab (*Ztschr. f. Augenh.* 2: 235, 1899), who described it as "alphabet keratitis," beginning at puberty and always being bilateral. In this condition the opacity consists of many fine lines arranged in a lattice-like pattern. Dimmer (*Ztschr. f. Augenh.* 2: 353, 1899) stated the belief that recurrent inflammation is present early in the condition, but Parsons (The Pathology of the Eye, New York, G. P. Putnam's Sons, 1904) did not concur in this opinion.

Numerous theories have been offered to explain the origin of corneal dystrophy. In reviewing the literature one is struck by the number of cases occurring in members of the same family. The most noteworthy example of familial occurrence is Frykholm's (*Klin. Monatsbl. f. Augenh.* 94: 76, 1935) series, in which 18 of 42 members of a family were affected, over 6 generations. Judd (*Am. J. Ophth.* 16: 310, 1933) reported 9 cases in 3 generations, and M. Freiberger (Corneal Dystrophy in Three Generations, ARCH. OPHTH. 16: 257 [Aug.] 1936) reported 12 cases in 9 generations. On the basis of his series, Freiberger called the condition "familial corneal dystrophy." This term should not be adopted, for in a large number of the reported cases the condition was present in only 1 member of a family, with no history suggesting that other members were involved. The authors of the three series just cited consider that the condition is inherited as a dominant trait and is not sex-linked. It occurred with equal frequency among the male and female members of the families and was transmitted through both sexes.

Fischer (*Arch. f. Augenh.* 98: 41, 1928; 100-101: 480, 1929) has demonstrated a close relationship between the transparency of the cornea and its water content. If the cornea loses or takes up more than about 20 to 30 per cent of its water content it becomes semitranslucent. This author has also shown that the imbibition of water is related to the integrity of the epithelium and endothelium and that if either layer is injured the cornea becomes permeable to water in both directions. He has observed that the intact cornea maintains an irreciprocal permeability toward water and sodium chloride and that the unidirectional flow is toward the aqueous. However, Krause (The Biochemistry of the Eye, Baltimore, Johns Hopkins Press, 1934) expressed the belief that the epithelial permeability is evidently influenced through the action of the trigeminal nerve. Tagawa (*Arch. f. Augenh.* 102: 231, 1929) has found that the water content of the epithelium was regulated by the nerves and that after irritation of nerves the amount of water in the cells was increased. Also, after sectioning of the trigeminal nerve the epithelium swelled, and islands of cells were desquamated.

On the basis of this work one can offer the hypothesis that the initial lesion in corneal dystrophy is an irritative lesion in the corneal nerves. This results in increased permeability of the epithelium, with an increase in the water content of the epithelial cells and superficial lamellae and a change from irreciprocal to reciprocal corneal permeability. This

brings about a disturbance in the oxygen metabolism, resulting in necrosis and hyalinization of those cells which are farthest removed from the limbus and aqueous, that is, the cells in the superficial layers of the center of the cornea, as is seen in this type of corneal dystrophy.

In summary, the following points are noted:

1. A review of the literature of the nodular and the lattice type of corneal dystrophy leads to the belief that they are merely variations of the same condition.

2. Although the variety of the pathologic changes is confusing, the frequency with which hyaline masses are seen in the epithelium and superficial lamellae would permit this nodular formation to be called the characteristic lesion.

3. The theories offered in explanation of corneal dystrophy are discussed and found to be unsatisfactory.

4. An explanation of the condition is offered, based on the disturbed permeability of the cornea resulting from an irritative lesion of the corneal nerves.

DISCUSSION

DR. MARK J. SCHOENBERG, New York: Very stimulating and interesting is Dr. Rones' attempt to formulate a hypothesis as to the manner in which the nodular lesions of the cornea are formed. Fischer, Tagawa and Krause have contended that the imbibition of water by the corneal tissue is related to the integrity of the epithelial and endothelial layers, that the permeability of the cornea is influenced by the action of the trigeminal nerve, that the water content of the epithelium is regulated by the nerves and that after irritation of nerves the amount of water in the cells is increased. On the basis of the findings of Fischer, Tagawa and Krause, Dr. Rones has advanced the hypothesis that the initial lesion in corneal dystrophy is an irritative lesion in the corneal nerves, which irritation results in increased permeability of the epithelium, with an increase of the water content of the epithelial cells and superficial lamellae. This is followed by a disturbance of the oxygen metabolism, with the formation of nodules and necrosis and hyalinization.

SECONDARY CATARACT, WITH PARTICULAR REFERENCE TO TRANSPARENT GLOBULAR BODIES. DR. ALFRED COWAN and DR. WILFRED E. FRY, Philadelphia.

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USE OF CONCENTRATED PREPARATIONS OF EPINEPHRINE IN GLAUCOMA, IRITIS AND RELATED CONDITIONS. DR. MEYER WIENER and DR. BENNETT Y. ALVIS, St. Louis.

The use of strong preparations of epinephrine over a period of years in 13 cases was recorded. This experience and the rich literature on the subject make possible certain conclusions which constitute a fairly comprehensive set of working rules for the use of this important therapeutic agent.

1. A concentrated solution of epinephrine hydrochloride is a powerful mydriatic capable of lowering the ocular tension, producing an acute rise of the general tension and causing severe pain and, occasionally, general symptoms; hence, when it is first used conditions should be such that the patient can be observed for one or more hours.

2. Epinephrine is a valuable aid in cases of chronic simple glaucoma in which miotics must be used for prolonged periods in lieu of the preferred surgical measures.

3. Chronic secondary glaucoma generally does not yield to epinephrine, but may do so, and a trial is justified.

4. After operation for glaucoma, epinephrine may be effective in bringing about a state in which the tension remains normal for long periods without treatment.

5. Glaucoma, after needling of cataract or the membrane of an after-cataract, has been controlled in a large percentage of cases by epinephrine, given with or without miotics.

6. Epinephrine is invaluable in breaking up adhesions in cases of iritis and uveitis.

7. The most convenient form for office and prescription use is an ointment in a water-soluble base (tragacanth jelly) or in an oily base (petrolatum and hydrous wool fat).

DISCUSSION

Dr. E. C. Ellett, Memphis, Tenn.: Ophthalmologists have gone a long way in the knowledge and application of epinephrine since Darier first introduced it in the field of ophthalmology in 1896 as an agent to dilate the pupil and reduce the intra-ocular tension. The present manner of employing it may be said to date from 1922, when Carl Hamburger advocated the use of epinephrine in the treatment of simple glaucoma, later developing the preparation which he called glaucosan (dextro-glaucosan). This is a combination of dextrorotatory epinephrine and methylamino-acetocatechol. This was intended for subconjunctival injection. .

Continuing his researches, he developed a mixture of levorotatory epinephrine and methylamino-acetocatechol, which he called levoglaucosan. This proved to be active enough to use by instillation only. It causes dilatation of the pupil and a decrease in the intra-ocular tension.

The method of instilling levoglaucosan is simple. The eye is anesthetized with phenacaine hydrochloride or butyn, and then, with the patient in a recumbent position, 2 drops of the solution as furnished in the ampules is instilled into the cul-de-sac. The patient is directed to rotate his globe in the four cardinal directions while the lids are held open, which allows the drug to come into free contact with the globe. Drops are instilled again from three to five times at intervals of fifteen minutes. In a few moments after the first drop is instilled the sclera should become porcelain white, the aperture of the lid widen and the pupil dilate regularly, while a zone of ischemia usually develops on the lids, cheek and temple. Practically always there is a distinct drop in the tension. Sometimes the cornea appears as though glistening powder had been sprinkled on it. This appearance soon passes off.

There has recently been available, due to the efforts of Dr. John Green, a synthetic epinephrine bitartrate, dispensed as a powder in glass ampules. Each ampule contains 0.091 Gm. of the drug, and when dissolved in 2.5 cc. of water this makes a 2 per cent solution. At the suggestion of Dr. Melville Black, my associates and I have used this in the powdered form, taking up a small amount on a very small eye curet. The solution is not stable, but the powder is, and the use of the powder is satisfactory and economical.

As regards these preparations of epinephrine, my associates and I can speak favorably of their use in iritis, especially in those troublesome cases in which the tension is increased. The pupil can be widely dilated, adhesions broken and the tension usually reduced, at least, never elevated. The dilatation can often, but not always, be maintained with atropine. Subsequent applications of epinephrine seem much less effective than the first one. I have published my observations, with reports of many illustrative cases, to which I could add many more. The effect in breaking adhesions and fully dilating a rebellious pupil is striking, and the pain, when present, is usually relieved.

Without apparent cause the application is sometimes painful. My associates and I administer the drug with the patient lying down, and first give 10 grains (0.65 Gm.) of acetylsalicylic acid. In addition, some opiate should be at hand for hypodermic injection. The unpleasant effect does not interfere with the dilatation of the pupil, and it is not due to hypertension.

In glaucoma the results of the use of epinephrine are not so good. Sometimes, but not always, the tension in simple glaucoma can be reduced by this drug and can subsequently be kept under better control with miotics, but the result is not constant; at best, epinephrine is only an adjuvant to miotics, and sometimes it will precipitate a severe attack of acute glaucoma. The form which was introduced as especially effective in acute glaucoma, namely, a 10 per cent solution of histamine, has proved to be a flat failure, in my experience. It neither contracts the pupil nor lowers the tension.

NAEVUS FLAMMEUS AND GLAUCOMA. DR. K. L. STOLL, Cincinnati.

This article will be published in full later.

SCLEROMALACIA: REPORT OF A CASE. DR. FREDERICK A. KIEHLE, Portland, Ore.

Rather recently, van der Hoeve (abstr., *Zentralbl. f. d. ges. Ophth.* 26: 504, 1932) reported several cases of scleral disease under the title of scleromalacia perforans, which fall under a classification that Rochat (abstr., *Zentralbl. f. d. ges. Ophth.* 29: 663 and 664, 1933) had termed scleritis necroticans. Van der Hoeve made the suggestion that this condition may be more frequent than is commonly suspected and emphasized its association with the severe polyarthritis present in the same patient.

The case to be briefly described here is one of interest as further emphasizing this connection. It is unusual in that the condition falls into none of the known classifications of scleral diseases and varies in certain particulars from the condition in van der Hoeve's cases, in which per-

foration occurred. No condition resembling it can be discovered in reports in the literature, nor has a case of any similar condition been encountered in the experience of several distinguished confrères who have examined the patient.

Since the case is evidently one of degeneration of the sclera, the term scleromalacia (Professor van der Hoeve's nomenclature) seems most appropriate.

The patient, a woman, is now 64 years old. She has been a victim of rheumatism since the age of 21. She took to her bed nine years ago because of a fractured hip, and has been there continuously since because of deforming polyarthritis. Practically all the joints are affected. Her hands are gnarled, her feet less so. Her spine is nearly rigid. There is some movement of the knees, elbows and shoulders, and she can stand and even take a few steps if aided. Tophi are present in several joints.

In 1929 both eyes showed similar changes, which were more marked in the left eye. The first glance seemed to reveal a huge anterior staphyloma, though a moment's examination showed the fallacy of this observation, as all the bulbar measurements were normal. The unusual appearance was due to the color of the sclera, which had lost its normal whiteness and was slate gray. At a distance the sclera resembled the cornea with the gray iris behind it. The conjunctiva appeared to be intact. The episcleral tissue throughout, after a stage of edematous swelling, has, with a gradually receding margin, been completely absorbed.

When first seen, the cornea and the other media were clear and permitted satisfactory intra-ocular examination. Such examination revealed no abnormality of the fundus. The tension has never been elevated. As the years passed other signs of degeneration occurred— both lenses are now cataractous, and both eyes are sightless except for perception of light. The left cornea has in recent years shown generalized clouding and roughening but without ulceration. I suspect that uveitis has existed, but it has been so insidious as to cause no symptoms. A recent development is the occurrence in each iris of multiple radiating perforations, two or three in line, each perforation measuring from 1 to 3 by 1 mm. These radiating lines are separated by distances of 3 or 4 mm. around each iris. These perforations appear to be ruptures through the thin, atrophic iris.

At no time has pain been an important feature of the condition. Such discomfort as the patient has suffered was described rather as "burning" and "irritation." As to the inception of the ocular symptoms, her recollection is that the trouble in the eyes began with the common sensation of a foreign body in the eye. There is no history of trauma or of previous iridocyclitis. The palpebral conjunctiva appears normal, as do the lids and other adnexa. Bulbar movements are normal. Thorough general physical examination by a competent internist revealed no systemic disease apart from the chronic arthritis. Questions as to the patient's customary diet suggested no vitamin deficiency.

It is significant that all conditions of this type involving degeneration of the sclera have occurred in patients suffering from arthritis deformans. It is suggested that defective metabolism in the fibrous tissue and insufficient elimination of waste are a possible basis of arthritis deformans. This may be a factor in scleromalacia. Friedenwald

(*Am. J. Ophth.* 4: 433, 1921) stated that arthritis deformans is regarded as a chronic infection. He, with a number of other writers, quoted Osler (The Principles and Practice of Medicine, ed. 11, New York, D. Appleton & Company, 1920, p. 1160): "Arthritis deformans is regarded as a chronic infection caused by certain varieties of streptococci and secondary to a focus of infection somewhere."

DISCUSSION

DR. WILLIAM L. BENEDICT, Rochester, Minn.: I saw the patient in April of this year with the condition as depicted on the screen. It seemed to me that not only was the sclera undergoing a gradual process of absorption but that in certain areas there was an infiltration which could not be looked on as inflammatory but was probably a condition that one frequently sees in cases of malacia, in which there is a deposit of fat and other abnormal tissues in the region.

Furthermore, through the very thin sclera and through the nearly transparent conjunctiva appeared the atrophic uveal tract. The slate color of the eyeball evidently was due to the pigment in the uveal tissue showing through. The mottled appearance was due to the infiltration of the region which once had been occupied by the sclera.

It was striking to note the marked changes in the sclera associated with such an insignificant change in the cornea. For many years after the scleral degeneration had been progressive the cornea had remained nearly clear, even in its marginal portions.

One recognizes well the changes that occur in the choroid in cases of inflammatory diseases of the sclera and in those of scleritis and episcleritis. I consider the change of the choroid, the spaces in the iris and the opacity in the vitreous as secondary to the disease in the choroid and not necessarily a part of the process in which the sclera was the most prominent tissue affected.

The relationship between the disease of the eye and the arthritis or fibrositis is more than incidental. Evidently the inflammatory process had progressed much further in the joints and in the muscles, and evidently some changes in the bones had taken place. I can only agree with Dr. Kiehle that both the changes in the eye and those of the bones must be secondary to some primary cause.

GERMAN OPHTHALMOLOGICAL SOCIETY

Fifty-First Annual Meeting, Heidelberg, July 6-8, 1936

DOCENT DR. M. BÜCKLERS, Tübingen, *Reporter*

TRANSLATION BY PERCY FRIDENBERG, M.D., New York

Opening Session

At the beginning of the session the Dean of the Medical Faculty announced that the University of Heidelberg, in connection with its Five-Hundredth Anniversary Jubilee, had conferred the degree of honorary doctor on Professor Van der Hoeve, of Leyden, Netherlands.

This honor, of which Professor van der Hoeve expressed his appreciation, met with marked and general approval. The presiding officer, Professor Löhlein, then welcomed the visiting colleagues who were present in large numbers from abroad, as well as from other cities of the Reich. He called attention to the necessity and importance of the experience and daily detailed work of the practicing ophthalmologist, as well as the experimental work and laboratory data of the research worker. Many questions remain to be answered, as shown, among other things, by the wealth of data in regard to hereditary diseases of the eye collected by ophthalmologists all over the world to a degree unequaled in any other specialty and so greatly to be envied. Since the prevention of hereditarily diseased offspring and descendants has become a matter of legal regulation and an old requisite of preventive medicine has been fulfilled, it is necessary to take stock and see to what extent ophthalmologists are standing on sure ground and what problems, on the other hand, still await their solution.

The president then extended the thanks of the society to the official reporters, Drs. Oehlkers, von Verschuer, Waardenburg and Clausen, for their cooperation in preparing and presenting the fundamental facts on this subject.

First Scientific Session

Monday, July 6, 1936, 8 a. m.

J. VAN DER HOEVE, Leyden, Netherlands, *Chairman*

SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY: I. HEREDITY FROM THE GENERAL BIOLOGIC STANDPOINT. DR. F. OEHLKERS, FREIBURG IN BREISGAU.

Under certain circumstances the general science ophthalmology is obliged to elaborate material which is not significant or important from the practical standpoint, in order to get and guarantee theoretical progress as quickly as possible. This drawback can be partially overcome or at least neutralized to some extent by cooperation with physicians and breeders. To characterize the present status of research in heredity, three fields have been chosen. The first is concerned with the chromosome theory. The proofs of this theory start with the mendelian law and show, to begin with, that the mechanism of division of the cell nucleus at maturity guarantees the recombination of the rudimentary units postulated by Mendel's law. Then Morgan's theory of linear arrangement of the genes in the chromosome was discussed in line with recent observations and experimental research on anomalies of chromosomes and roentgen ray translocation and was shown to be correct as indicated on chromosome charts. On the other hand, the hypothetic distances between the genes, estimated from the "crossing-over" values, do not correspond to the data of the latest investigations. Accordingly, the chromosome charts are to be considered as purely graphic representations of the crossing-over values. The significance of the chromosome theory for the construction of a theory as to the genetic condition of a population was then considered. If this condition is connected with the parents by way of division of the cell nucleus at maturity and sexual propagation, one must expect basic variation in the hereditary

characteristics of successive generations in case of a heterozygotic foundation. If, on the other hand, descendance is connected with the starting organism merely by way of the usual mitotic division, both links of the chain are equal from the standpoint of genetics. The question of development of the hereditary anlage was then considered in the light of latter day research. Recent experiments with mosses (von Wettstein) seem to show that a dominant and a recessive quality, respectively, depend in many instances on the quantity of genes and that alteration of this factor may bring about a reversal of type. There was a short reference to hereditary variation, the frequency of appearance of new hereditary rudiments and the possibility of increasing the speed of mutation by such agencies as heat and the roentgen rays.

II. Inheritance of Disease from the General Medical Standpoint. Dr. O. von Verschuer, Frankfort-on-Main.

What rôle, if any, is played by a hereditary pathologic tendency in the development of a disease? One must, first, put in a separate division those diseases which are, without exception, of hereditary provenance. The conditions are determined with much greater difficulty in the case of diseases which may occur as hereditary and nonhereditary forms, respectively, with the same or a similar clinical picture.

The differential diagnosis of these forms is of great practical importance, and further working out of the knowledge of this clinical factor is needed. The second question involves the construction of the genotype. Besides the theoretical questions previously considered to be of a special biogenetic bearing, one must consider as of most practical importance a decision as to whether the hereditary predisposition to a given disease is specific or nonspecific. The third question is that of the relative significance of inheritance and environment as etiologic factors in disease. A few examples were adduced to show that the conception of the relation of heredity and environment is to be understood variously for different diseases and for nosologic reasons. The final question, that of heredoprognosis, depends largely on the more important considerations of heredodiagnosis and requires continued and more exact research into the actual hereditary conditions of families.

III. Heredity in Ocular Disease. Dr. P. J. Waardenburg, Arnhem, Netherlands.

Discussing the data derived from research on twins, the reader suggested, on account of their brevity and accuracy, the terms gonosomal and autosomal, respectively, for rudiments (anlagen) connected with X-chromosomes, on the one hand, and those connected with the other chromosomes, on the other. In ophthalmology, serial investigations which are continuous, without gaps and requiring no rejections, are far behind the case reports in number. However, the mode of investigation by study of individual cases is of practical value in corroborating the insight into the nature of the hereditary basis, afforded by other methods, of many autosomal and gonosomal ocular characteristics. As examples of variation in manifestations in enzygotic twins the reader instanced a decidedly frequent discordance of amblyopia with concordant strabismus, reported by numerous authors; concordance with an eight

year interval in severe tuberculosis of the iris (van der Hoeve); concordance, together with discordance, in syphilitic conditions of the eye (Vogt, Glatzel); marked difference in degree of ablatio retinae pellucida (Weve), and concordance of the length of the base of the skull and the sphenoidal angle in cases of steeple skull and craniofacial dysostosis, with variation in other dimensions of the skull (Waardenburg). Examples of conditions with far reaching concordance in enzygotic twins are exophthalmic goiter; unilateral as well as bilateral hydrophthalmos; homolateral glioma, senile glaucoma, Leber's disease (in affected persons as well as in transmitters) and atypical forms of pigmentary degeneration of the retina and/or choroid. The total refraction, the corneal refraction, astigmatism, epicanthus and strabismus were considered more from the standpoint of mass statistics. Recently, a hereditary basis has been found for many senile changes (Vogt). Many valuable facts were brought out in regard to the very slight nonhereditary spread (*Streuung*) of refraction. Disturbances of color sensation are as yet provisionally attributed to multiple gonosomal rudiments (*Allele*) at various points (loci). Megalocornea, bulbar albinism, "extra-ocular" nystagmus, night blindness associated with myopia, pigmentary atrophy of the retina and Leber's disease were discussed. Among the diseases described only once were: gyrate atrophy; a form of microphthalmos; a form of myopia; a hypoplasia of the stroma of the iris associated with secondary glaucoma; a sibship or kinship with macular degeneration; a similar relationship with night blindness, myopia, amblyopia and nystagmus, and probably a certain form of cataract. In line with these are some diseases connected with disease of the skin or nerves. The various forms of a dominant gonosomal anlage, nystagmus and Leber's disease were discussed and analyzed; and in connection with the last-mentioned condition Lossen's rule, which has a practical bearing in giving counsel on the question of marriage, was discussed. There are probably a number of other more or less regularly dominant gonosomal ocular characteristics. With some caution one may include flocculi iridis and some manifestations in the group of disturbances of ocular motility, of cataracts and of pigmentary degeneration of the retina and choroid, as well as some rather rare variations (polycythemia; atrophy of the optic nerve associated with diplegia and feeblemindedness, and other conditions).

In discussing the dominant autosomal anlagens, the reader presented a series of "harmless" characteristics, but took exception to the idea that all dominant characteristics represent slight variations. A number of corneal and lenticular opacities, the coloboma group and glaucoma are not such variations. Dominant variations involving the organ of vision, *sensu strictiori,* i. e., the eye and the optic nerve, serious conditions threatening blindness and destruction of the globe, include glioma (exceptionally), detachment of the retina, pigmentary degeneration of the retina, macular degeneration and infantile atrophy of the optic nerve. Serious irregularly dominant diseases include angiomatosis, steeple skull and other anomalies. Most pathologic manifestations in the field of ocular motility and of the lacrimal apparatus are dominant. Definite knowledge of the heredoprocess (*Erbgang*) in recessive autosomal anlages depends largely on a statistical accumulation of data gained from a. study of marriages of persons related by blood. Such

was the case with pigmentary atrophy of the retina, albinism, achromatopsia, hydrophthalmos, microphakia, hemeralopia of Oguchi and juvenile amaurotic idiocy. As recessive characteristics were noted a whole series of anomalies which may affect any ocular part except the lacrimal apparatus and the sclera. The most serious are the disorders of the organ of vision, the visual apparatus itself. The reader showed a number of pictures illustrating the spread of such disorders in regions where intermarriage is common (*Inzuchtgebieten*).

In conclusion, considering the difficulties in the way of accurate investigation of heredity in human beings, it would be of the greatest value if in the future more and more attention were paid to possible intermediary stigmas of heterozygotes with recessive characteristics, and if, as far as possible, a search were made for marriages of transmitters of characteristics in order to find out whether there are any differences as to the degree, extent and interval of time between heterozygotes and homozygotes. In the course of the report examples of change of dominance were cited, and some differences between the white and the yellow race (Japanese) were discussed.

PRELIMINARY ANNOUNCEMENTS AND DISCUSSION OF REPORTS I, II
 AND III. RESEARCH INVESTIGATION OF THE ORIGIN OF
 AMETROPIAS. DR. PAUL, Lüneburg.

According to Steiger's "mosaic" theory of heredity, anomalies of refraction of low or medium degree develop on a basis of fortuitous combinations of unsuitable and disparate optical systems and axial lengths. The reader had shown, as far back as 1935 (at a meeting of the Northwest German Ophthalmologic Society), that his studies of family trees did not support this theory. He presented statistical tables and graphs showing the result of investigation of family trees, which had become much more numerous of late, of families in which the corneal radius of each parent was known, the refraction determined and the axial length of the globe calculated. In one division were grouped those families in which both parents were emmetropic. There was a total of 215 families, with 377 examined offspring in the first filial generation. It appeared that in families in which there was a correspondence in the dimensional disposition (anlage) of the eyes of the father and the mother (104), 22 per cent of the progeny manifested myopia, and 9 per cent manifested a high degree of hyperopia. In families in which this dimensional correspondence between the paternal and the maternal eyes was missing (111), 19 per cent of the offspring were myopic, and 11 per cent had a rather high degree of hyperopia. There was no indication of an increased tendency to the appearance of anomalies of refraction in the filial generation of parents with dimensional noncorrespondence of the eyes, although this would have been expected according to the mosaic theory.

In those families (87) in which paternal generations had shown myopia, myopia was also found in the filial generation in 50 per cent. It is accordingly evident that the development of myopia in the children is influenced by the occurrence of that anomaly of refraction in the parents, and one must accept as a fact the inheritance of a tendency to the development of a certain form of the eye. Analysis of the individual family trees showed, however, that this developmental tendency

was not coupled with what one might call the dimensional heritage of the eye. The tendency to the development of a particular anomaly of refraction may well be attributed to a genetic factor quite different from the hereditary dimensional disposition of the eye. Wibaut has come to precisely the same conclusions from an entirely different order of investigations. In a future communication the reader intends to discuss the significance of the observation that, at least in youth, hyperopia appears to be more frequent in persons with a corneal refraction lower than normal, on the average, and myopia appears to be more frequent in persons with a corneal refraction higher than normal, and to analyze his material according to the principal biologic groups represented.

CORRELATION OF HEREDITARY DISEASE OF THE EYE AND OF THE CENTRAL NERVOUS SYSTEM. DR. BEST, Dresden.

As the eye may be considered as a portion of the brain protruded, one would expect numerous points of relation between the hereditary diseases of these two organs. However, their number shrinks markedly if one applies the conception of correlation in its stricter sense. An ocular disease may be secondary, dependent on a disease of the brain. Again, one may be dealing with localizations of one and the same disease in the eye and the brain, so that one infers the presence not of genotypically different diseases but of phenotypically differing manifestations of the same disease genus. As an example of dependent, secondary disease of the eye one might mention atrophy of the optic nerve in association with steeple skull, acrocephaly or Crouzon's disease. In cases of such association the atrophy of the optic nerve is the result of a previous papilledema. If, on the other hand, one attributes atrophy of the optic nerve to mechanical pressure on the nerve in its passage through the bony canal, due to an unusually low level of the middle fossa, one is dealing, more than ever, with a secondary symptom and not with a correlation. As examples many other analogous syndromes might be mentioned, such as oculomotor palsies in association with basal aneurysms with a congenital anlage, and atrophy of the optic nerve due to pressure of a dilated third ventricle in association with congenital hydrocephalus. Again, one cannot very well speak of a correlation in the genetic sense when disturbances of ocular function are associated with a neuropathy on account of the character of the cerebral localization, e. g., sympathetic pupillary paralysis or paralysis of the abducens nerve in status dysraphicus.

Finally, even when the same hereditary disease befalls the eye and the brain one is not dealing with a true correlation, *sensu strictiori*. This category takes in, primarily, neurofibromatosis, tuberous sclerosis and von Hippel-Landau's disease, as well as Hand-Christian-Schüller's disease, in which the same xanthomatous proliferations are found in the bones of the skull, the orbit, the hypophysis and even the retina. Aside from the aforementioned neoplastic formations, congenital malformations or developmental anomalies of an almost identical character occur in the eye and the brain, as in the clinical picture described by Schiötz as the vascular encephalotrigeminal syndrome. This consists in tortuosity of the retinal vessels or telangiectases of the trigeminal area associated with capillary angiomas in the brain. An example of pseudocorrelation in the motor field is chronic progressive ophthalmoplegia as a heredo-

degenerative disease. According to Marburg, this is probably an analog of progressive spinal muscular atrophy and of progressive bulbar paralysis and presents a unitary disease of the motor system (amyotrophia muscularis progressiva). To sum up these observations, in many instances of associated lesions of the eye and the brain there is no true correlation in the genetic sense. Considering true correlation of hereditary disease of the eye and the brain, this may be noted in one and the same patient or in different members of the same family. The latter form of correlation, for example, the occurrence in a subject of tapetoretinal degeneration while sisters and/or brothers are feebleminded, is perhaps at least as frequent as is correlation in one and the same person. It is not feasible to discuss all correlations which can occur, but one might mention briefly those cases in which correlation does not occur. In this respect, glioma of the retina is remarkable. Children with glioma of the retina—which is undoubtedly hereditary, once it has developed on a basis of mutation—are, as a rule, in all other respects uniformly normal physically as well as mentally in spite of the fact that there is a marked increase in the persistence force (*Durchschlagskraft*) in comparison with the immediate forbear, as indicated by the predominant bilaterality of this disease in the second generation as against a marked preponderance of unilaterality in the first. Conversely, glioma of the brain is rarely, if ever, associated with hereditary disease. Of diseases with correlated conditions may be noted especially feeblemindedness and idiocy, not so much on account of the various associated ocular developmental defects, e. g., errors of refraction, anomalies of structure of the nerve head, a persistent pupillary membrane and concomitant strabismus, but rather because they enable one to posit some basic principles. To begin with, it is worth mentioning that there is no definitely positive correlation on which one can count to any extent. As far back as the end of the last century the older physicians were forced to note how slight a value attached to what they then called stigmas of degeneration, if one wished to draw conclusions from them in reverse as to the condition of the brain.

It may be worth mentioning that in cases of feeblemindedness there is a tendency toward anomalies of the disk, tapetoretinal degeneration and a high degree of hypermetropia, i. e., anomalies in the form and structure of the globe, while degenerative-motor diseases, such as diplegia, are more frequently associated with concomitant squint. One thing is certain: The more marked the mental degeneration, the more frequent and intensive the correlated developmental anomalies of the eye. This appears especially definitely when one instances the true malformations of the brain, such as microcephaly, microgyria and macrogyria, and anencephaly. In these conditions the associated abiogenesis of the eye takes such marked forms as coloboma and analogous defects, aniridia, microphthalmos and anophthalmos. But here again the question arises whether even in such conditions one is dealing with an actual combination of dissimilar genes, with a phenotypically similar manifestation of one and the same disease genus in the eye and the brain or, finally, with a direct and immediate dependence of the ocular malformation on that of the brain. Similar considerations, by the way, apply to the frequent association of high degrees of hypermetropia and a short corneal radius (Kurtz, Vogt) with feeble-

mindedness and microcephaly, in which cases one may infer that a too small anlage in the brain is mirrored by a similar developmental discrepancy involving the eye.

The more one busies oneself with the subject of correlation, the more one realizes that there is still much to be learned before factual material can be grasped and reconciled with the calculations and the comparatively rigid concepts of genetics. In all probability one must take for granted a rather high degree of lability of genes which have arisen or been altered as a result of mutation.

FAMILIAL MACULAR DEGENERATION.   DR. GASTEIGER, Frankfort-on-Main.

A survey of 11 cases of familial macular degeneration noted in one family within three generations, with the family tree, was presented. Its earliest occurrence was at the age of 17, and the first indications consisted in slight irregularities of pigmentation at the posterior pole, which later developed into delicate light gray flecks, resulting finally in a large dirty gray area. The successive stages could be observed in different members of the family. No other disturbances were noted, and, in particular, no mental deterioration was observed, in the family. Vision in the initial stage was 5/10 = 5/5, and it decreased to 1/50 = 2/50, when marked ophthalmoscopic changes were noted. The reader then reported a case of congenital oculomotor palsy, showing pictures illustrating an associated ptosis. Motility was completely abolished except in a single case in which the left eye showed a slight abduction. In one member of the family there was, coincidentally, a coloboma of the nerve entrance in one eye; besides, nystagmus and astigmatism were noted. The anomaly was checked up positively through three generations, in one of which all the members of the family, without exception, were affected, according to the statements of those members who were examined. Relatives assured the investigator that the disease had also been present in many earlier generations. As the family was of foreign origin, detailed and intensive investigation met with serious obstacles. A dominant heredity may definitely be accepted.

SIGNIFICANCE OF PERCENTAL CALCULATION OF THE DIMENSIONS OF THE EYEBALL IN GENETIC RESEARCH.   DR. PAUL, Lüneburg.

The size of the globe and that of its component parts is influenced by age, sex, race and other factors. In order to test these dimensional values correctly and to judge the relation of different eyes to one another, they must always be considered with reference to a normal or average eyeball. This will be facilitated by a representation of the dimensional values of such an eyeball in percental terms. An emmetropic eye corresponding in form with the usual schematic eye—which may be called the original or eo-eye (*Urauge*)—can be calculated percentally as a pure quantitative variation of the latter. In the case of anomaly in form, the calculation must make use of a similarly anomalous formally schematic eye, the "secondary" eye. The corneal radius of the secondary eye is put down as corresponding with the dimension of the original eye (*Urauge*). The axial length of the secondary eye can be calculated as a percentage of the "original" axis, and this value can be applied as an indication of the activity of the lenticular system—

designated as the lens factor (*Linsenwertigkeit*)—which is coupled with it, as both the original and the secondary eyeball are conceived of as emmetropic. From this value it is possible to determine the accommodative power and the position of the lens system, with the use of the formulas which the reader has worked out and which he reported in a previous communication.

Any emmetropic eyeball shows a dimensional variation when compared with a corresponding, suitable secondary eyeball. In case of ametropia a tertiary eyeball is used as the schematic eye. Its axial length is a percentage of the axial length of the secondary eyeball and, as it is coupled with the error in refraction, can be applied to indicate and measure the latter. Indirectly, by means of the tertiary (and the secondary) eyeball, the corneal radius and the axial length of any eye can be calculated in percental terms of those of the original eye, and the refraction of the lens can be calculated from the lens factor (*Linsenwertigkeit*). The eo-eye, or "original" eye (*Urauge*), is not always suitable as a standard of comparison. However, the comparative percental values found by comparison with an "original" eye can be corrected to comparative values in reference to a more suitable test, or comparison, eyeball, by dividing the figure by the corresponding variations of the chosen standard of comparison. In this way one is enabled to express the dimensional values of the eye under investigation in a form which takes into consideration age, sex, race and other factors of its individuality. It is only by means of such a modification (*Umormung*) that the dimensional data for an eye gain practical significance and value for research in genetic statistics.

Calculations of the corneal radius in grown men (1,002) and women (1,334) from the reader's collection of family trees showed that on the average the corneal radius was 1.7 per cent greater in men than in women. The variation polygon of the corneal radii of men and women, respectively, has, as is well known, a different position and a different form. A combined variation polygon constructed from the figures of the eyes of males and females resembles rather a polygon bounded by a binominal curve, it is true; but, as shown by graphic demonstration and illustrations, it is determined absolutely by the fortuitous combination of the eyes of the two sexes. This indecision as to the exact figures disappears completely if for absolute values one substitutes percental comparative figures calibrated with reference to a suitable standard eyeball.

THE THREE FORMS OF FAMILIAL CORNEAL DEGENERATION AND THEIR HEREDITARY TRANSMISSION. DR. M. BÜCKLERS, Tübingen.

The research, carried out in association with cand. med. Gilch, took in 35 localities and 800 persons. The most frequent form of hereditary corneal dystrophy is characterized by glassy crumbly deposits (*Bröckelchen*) which are sharply defined and surrounded by clear corneal tissue (*Fleischer*). This "crumbly" type of dystrophy begins in the fifth year of life in the form of extremely fine radiating lines and progresses slowly in extent and depth, filling a disciform area between 30 and 40 years of age, which later becomes still more dense and wide. The extreme periphery always remains clear. Two family trees, with 68 members affected altogether, show simple dominance.

The other type of dystrophy, which has also been described as "nodular" by Groenouw, also begins in the first decade but with a delicate, diffuse clouding of the entire corneal surface. Very soon flecks appear, which are superficial as far as the middle of the cornea is concerned but which lie deep in the periphery. In later life and old age the cornea is filled with so many dense opacities of this punctate, flecked form (*Fehr*) that the subject is practically blind. This type is recessive. In the lattice-like (*gitterig*) type of dystrophy, which Haab described, there appears rather early the well known system of characteristic double-contoured lines which bifurcate toward the summit of the cornea. These lines correspond to fine clefts in the anterior layers of the stroma, and this causes a peculiar glassy aspect in reflected (*rück-fallend*) light. Scattered among these lines are noted nodular thickenings which at various points raise up the epithelium, as in the flecky, punctate type of dystrophy. The family trees showing instances of authentic and undoubted lattice-shaped corneal dystrophy thus far collected reveal a dominant heritage. According to the reader of the paper, a transition from one of these fundamental corneal dystrophies to the other has never been observed. Accordingly, the inclusive term familial corneal degeneration should be dropped.

A FAMILY WITH CONGENITAL MACULAR DEGENERATION. DR. JUNG, Giessen.

A family in which 22 cases of macular degeneration are reported was studied for the first time by Vossius at Giessen as long ago as 1880. The cause of the macular degeneration was not syphilis, as was formerly assumed, but a heredity which is expressed in a decidedly intensified degree by the inbreeding which was widespread in this family. There are a number of clinical details speaking strongly in favor of this theory as to the etiology. These are: first, the congenital nature of the disease; then, the clinical findings, which are always the same, and the uniform localization of the lesions below the fovea; furthermore, the almost exclusively bilateral occurrence, and, finally, the noteworthy fact that the disease occurs in two separate sibships (*Sippen*), for which, however, a common origin was proved. The reader's investigations of 300 persons, far exceeding in number all those studied in previous researches, resulted in the addition of 8 cases to the 14 already reported in the medical literature. In this connection the cumulative occurrence of other developmental defects of the eye, mainly anomalies of refraction, is striking. For instance, hyperopic astigmatism was found in 68 per cent of the cases of macular degeneration; it was present in 10 per cent of the other cases as well. Another interesting feature is the way in which amblyopia occurs intermittently in cases of macular degeneration. The latter disease does not, however, follow the laws for a simple recessive heritage, as has been generally accepted, according to Rieger, nor can one agree with Behr, who assumes an anlage which up to then had been inherited recessively. A stronger claim to correctness is recognized in Rieger's theory, which accepts a dominant factor that assumes a hypostatic behavior toward another dominant factor of a second gene, so that the latter acquires the significance of an inhibitory factor. The reader is inclined to accept Siemens' hypothesis of an irregular dominance.

(To be Continued)

Book Reviews

Studies on the Aetiology and Pathogenesis of Cataract Zonularis: An Academic Treatise. By Gunnar von Bahr. Pp. 236, with 4 plates. Uppsala: Almqvist & Wiksells, 1936.

This work on lamellar cataract is a scholarly addition to the excellent monographs which have recently been appearing in Sweden.

Part I is devoted to a review of the literature. Here one learns that, while zonular cataract was probably first described by Schmidt in 1800, it was Jaeger who with the ophthalmoscope first differentiated it from other congenital types of cataract as a form showing opacities in a deeper layer of lenticular fibers which enclosed a transparent nucleus and were themselves surrounded by a normal cortical layer. The diameter of the zonular cataract varies between 5 and 8 mm., and the opacity, which is composed of closely set, tiny dots, is from 0.5 to 0.7 mm. in thickness. There may be "riders." The outer boundary is always sharply defined; the inner boundary is less distinct. This type of cataract may be partial, and rarely double layers of cataract are seen. It may also be associated with a posterior polar, a nuclear or some other variety of cataract. It is usually found in children, the incidence varying from 0.09 to 1.45 per cent, and females are affected more often than males.

The etiology is obscure. Since the fetal lens has a diameter of about 5.75 mm. at birth, it is obvious that this opacity can form either before or after birth. Genealogical studies have shown that the condition may be inherited. Thus, of 443 members of affected families, there were 236 affected persons, many of whom also had defects of the enamel of the teeth and some of whom had suffered from "fits" during infancy. The association of zonular cataract with defects of the enamel was ascribed by Davidson (in 1865) to zonular deformities in two epithelial organs, the lens and the enamel of the permanent teeth.

A summary of the reports of 14 authors revealed that of 743 patients with lamellar cataract, 51 per cent had suffered from tetany, 52 per cent of 711 had hypoplasia of the enamel, and 46 per cent of 714 had rickets.

In his review of the experiments of other workers von Bahr notes that a rachitogenic diet will not cause lamellar cataract, that cataract due to tetany can occur in a person without manifest tetany, that a decrease in the calcium content of the aqueous may be a possible factor in the production of lamellar cataract and that tetany may cause hypoplasia of the enamel in man. Rickets and other general debilitating diseases probably predispose to defects of the enamel. If lamellar cataract has the same cause as hypoplasia of the enamel it must be formed by the end of twelve or eighteen months after birth.

In part II von Bahr gives a detailed description, with protocols and illustrations, of his experiments with rats. This part of the monograph will be of especial interest to research students, as the author used roentgenograms of the knee joint to determine whether rickets was present, and the galvanic current was used to determine alteration in

the neuromuscular excitability. He also examined the lenses both with the slit lamp and with the ophthalmoscope for the presence of opacities of the lens, and the incisor teeth were observed with a corneal microscope for irregularities in the enamel. Analysis of the blood was made a few times to determine the calcium and the inorganic phosphorus content, and a few lenses were examined histologically.

Part III contains the author's conclusions, and in part IV is a summary of his work, which may be briefly abstracted as follows: When young rats were fed a rachitogenic diet continuously, no cataract was formed, but when there was a change to fasting or to a normal diet, cataract often appeared. Rats made rachitic with Steenbock and Black's ration 2965 showed a more marked and prolonged hyperexcitability in reaction to the galvanic current than nonrachitic rats when the diet was changed to one rich in phosphates. The rachitic rats then often showed manifest signs of tetany. In rachitic rats with tetany, cataract generally developed, at least in all cases in which the anodal opening contraction occurred at less than 0.3 milliamperes, and cataract developed in both the manifest and the latent form of tetany. Hypocalcemia was apparently an essential factor for the production of cataract, and the cataract seemed to be similar to that obtained after parathyroprival tetany in rats, appearing first in the superficial layers of the cortex as fine dots and radial lines (as seen by reflected light). The dots were not directly subcapsular, and the capsular epithelium appeared to be unaltered. The earliest cataractous changes were reversible. Often the formation of opacities progressed even when the tetanic condition had disappeared. The lenticular changes appeared partly as vacuoles and partly as radial lines with fine ground glass-appearing zones of discontinuity which were probably due to shrinkage of lenticular fibers. The impaired cortical layers were covered by fresh lenticular fibers so that a zonular cataract was formed. It could not be proved that tetany in the mother caused zonular cataract in the offspring. In attempting to obtain a decrease in the calcium content of the aqueous, subcapsular dots were sometimes obtained, but it could not be proved that these were due to the decrease in the calcium content. The results of these experiments seem to have some relation to pathologic conditions in man.

This is a most satisfactory monograph, since it not only gives a comprehensive review of the literature (over four hundred references are cited) but also presents the results of careful experimental work which was done to obtain further insight into the factors which may be responsible for the production of zonular cataract.

W. F. DUGGAN.

Bulletin of the Ophthalmological Society of Egypt. Vol. XXIX. Thirty-Third Session, 1936. Pp. 248, with illustrations. Cairo: Imprimerie Misr, 1936.

This bulletin records the proceedings of the Ophthalmological Society of Egypt at its thirty-third session held under the presidency of Dr. M. A. El-Kattan on March 27 and 28, 1936, at the Memorial Ophthalmic Laboratory, Giza. The volume contains 248 pages. 28 devoted to the business of the society and 220 to scientific reports by

members. The reports are well illustrated and contain much information of value to ophthalmologists, particularly those interested in external diseases.

A symposium on the pathology and treatment of trachoma is contributed by R. P. Wilson, M. O. Rifai, I. A. Mohamed, Max Meyerhof, M. A. El-Bakly, M. A. H. Attiah and A. F. El-Tobgy, and F. Masoud. Wilson touches briefly on the changes which take place in the conjunctiva and cornea following trachomatous infection, illustrating each important pathologic change with photomicrographs. He states that "the evidence is growing that the earliest sign of trachoma is the presence of intracellular inclusions in the cells of the conjunctival epithelium, the so-called Prowazek-Halberstaedter bodies." Of distinct interest is his finding that the corneal changes of trachoma occur early and that by the time typical lesions have appeared in the palpebral conjunctiva invasion of the cornea with new vessels has already occurred.

I. A. Mohamed discusses the possibility of trachoma of the lacrimal apparatus and concludes that the question must remain in doubt until the virus has been demonstrated in the lacrimal epithelium or until true trachoma has been reproduced by inoculation of tissue of the lacrimal gland or the lacrimal sac. In a study of 16 inflamed glands, the formation of follicles was noted in 2, but in no instance was there definite evidence that the inflammation was trachomatous. After giving a review of the literature Mohamed states the belief that there is strong evidence for trachoma of the canaliculi. Of 405 lacrimal sacs removed from trachomatous patients studied at the Giza Memorial Ophthalmic Laboratory, 131 showed the formation of follicles. Histopathologic study alone, however, is considered insufficient to elucidate the question of trachomatous infection of the lacrimal sac. Seventeen photomicrographs illustrate this report.

The history of the treatment of trachoma in antiquity, and during the Middle Ages in Arabia is the subject of a report of exceptional interest contributed by Max Meyerhof. It appears that the Greeks and, to an even greater extent, the Arabs possessed considerable knowledge of trachoma. They recognized different forms of the disease and its communicability. The Arabs knew of the connection of trichiasis and pannus with trachoma, and their surgical treatment of the disease was not very different from that practiced today.

The treatment of trachoma is considered completely in the reports by El-Bakly and by Attiah and El-Tobgy. These reports are valuable, since it is only in Egypt that any extensive therapeutic tests of the various antitrachomatous agents have been made. Copper sulfate, chaulmoogra oil and expression operation are considered to be of proved value.

Except for one report on fatty degeneration in a trachomatous cornea, the remainder of the volume is devoted to nontrachomatous conditions. Included among the reports are "Iridectomy Technique Modified to Suit Iridotomy Cases," by M. A. El-Bakly; "Elschnig-Török-Stanculeanu's Intracapsular Extraction," by A. Y. Fahmy; "A Peculiar Case of Polycoria," by M. A. El-Bakly; "A Case of Traumatic Total Depigmentation of the Iris," by S. Kamel; "Ectopia Lentis," by M. A. El-Atawi; "Histopathology of a Case of Capsular Glaucoma,"

by A. Y. Fahmy, and "Studies of a Series of Cases of Sympathetic Ophthalmia," by I. A. Mohamed.

The volume closes with a report of the Committee for Arabic Ophthalmic Terms, in which a large number of ophthalmic words and phrases are translated into Arabic. After revision this work will probably appear in dictionary form.

PHILLIPS THYGESON.

Saggi di oftalmologia. By Prof. Q. Di Marzio and associates, vol. 8, Bologna, 1935 and 1936.

The Ophthalmologic Institute of the University of Bologna, directed by Prof. Quinino Di Marzio, has published the eighth volume of "Saggi di oftalmologia." This book is a collection of reprints of forty original articles written by Dr. Di Marzio and his pupils Caramazza, Puglisi, Duranti, Spinelli, Lugli, Filippi-Gabardi, Biozzi and De Petri.

The researches have a vast scope. The subjects include the comparative anatomy of the eye, the biomicroscopy of the lens and the vitreous, studies on tuberculosis of the different structures of the eyeball, roentgenologic investigation of the optic foramen, the bacteriology of the eye, angiotenometry, ocular syndromes due to lesions of the internal carotid artery and of the sympathetic system, intermittent exophthalmos, and observations on the therapy of various ocular lesions.

These articles show the reader the serious scientific interest of the authors and their thorough knowledge of the subject. The literature is well covered and is discussed in a manner showing the alert supervision of Professor Di Marzio in the work of his associates at the Ophthalmic Institute of the University of Bologna.

G. BONACCOLTO.

Senile Cataract: Methods of Operating. Third revised edition. By Dr. W. A. Fisher. Price, $2.50. Pp. 150, with 181 illustrations. Chicago: H. G. Adair Printing Co., 1937.

In this new edition the chapters by the late Professor Fuchs, I. Barraquer, H. T. Holland, J. W. Wright, A. van Lint and O. B. Nugent have been retained. Fisher describes again the operative technic that he formerly used, which is a modification of Holland's and Smith's methods, and in chapter 9 he describes his new method, which he believes lessens the number of cases of ruptured capsule and loss of vitreous.

This method consists in the use of a suction pump. The cup measures 3 by 4 mm. and after being applied to the capsule of the lens it is slowly raised or moved from side to side until the attachment of the lens to the suspensory ligament is torn; then the suction is released, and the cataract is extracted by the technic of Smith.

Fisher found the Hildreth ultraviolet lamp to be particularly helpful in operations for cataract, as it brings out the cortex and capsule with better distinctness than is gained with the usual sources of illumination.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66, Boulevard Saint-Michel, Paris (6e).
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

INTERNATIONAL OPHTHALMOLOGIC CONGRESS

Secretary: Dr. E. Marx, Costzeedijk 316 Rotterdam, Netherlands.
Place: Cairo. Time: Dec. 8-14, 1937.

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

President: Dr. A. F. MacCallan, 33, Welbeck St., London, W., England.

FOREIGN

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

GERMAN OPHTHALMOLOGICAL SOCIETY

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. T. E. Ashdown Carr, 34, Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3, Midan Soliman Pasha, Cairo.
Time: March 1938.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Time: July 8-10, 1937.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.

Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.

Place: Lindley'a 4, Warszawa.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.

Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm, Sweden.

Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.

Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.

Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.

Place: San Francisco. Time: June 13-17, 1938.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Lee W. Dean, Washington University Medical School, St. Louis.

Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

American Ophthalmological Society

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.

Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.

Place: Hot Springs, Va.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.

Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.

Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

New England Ophthalmological Society

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time:
8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Salt Lake City. Time: May 24-27, 1937.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. H. Klemptner, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, Virginia Mason Hospital, Seattle.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501 Seventh St., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each
month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each
month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.
Place: New Orleans. Time: Nov. 30-Dec. 1-3, 1937.

Southwestern Michigan Triological Society

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand
Rapids, Mich.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. C. W. Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all
members have served.
Secretary: Dr. Edna M. Reynolds, 227 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the
month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Bldg., Atlanta, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.
Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York City.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. Rosenberger, 221 Fifth St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514 Sixth St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary-Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Edwin W. Burton, University of Virginia, University.
Secretary-Treasurer: Dr. George G. Hankins, 202 Medical Arts Bldg., Newport News.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. Samuel T. Hubbard, 294 State St., Hackensack, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. G. Henry Mundt, 30 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Andrew Timberman, 21 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Claude S. Perry, 40 S. Third St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. A. W. Davidson, City National Bank Bldg., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 720 Medical-Professional Bldg., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Philip B. Green, Old National Bank Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto, Canada.
Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 Eye St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

ESSENTIAL PROGRESSIVE ATROPHY OF THE IRIS

REPORT OF A CASE

HUGH S. McKEOWN, M.D.

NEW YORK

It is recognized, as has been stated by several authors, that the actual etiology of true essential progressive atrophy of the iris has not been discovered. The object of this paper is to report another case of a patient with this condition, who has been under observation for four years.

Comparatively few cases of essential atrophy of the iris have been reported, and knowledge of the disease is therefore incomplete. It is possible that some of the earlier cases reported may have belonged to the group of atrophies of the iris caused by, according to Fuchs,[1] (1) long-continued or recurring inflammation, (2) increase of tension involving the blood vessels at the root of the iris, (3) traction on the iris or iridodialysis, (4) too thorough absorption of swelling lenticular material or (5) advanced age or an unrecognizable cause. This was noted by Barr,[2] in 1934, who further stated that essential atrophy is progressive, so that congenital anomalies, such as aniridia or colobomas, should be easily ruled out.

REPORT OF CASE

Miss A. C., a graduate nurse aged 29, was first seen in September 1933, when she was referred for examination of her eyes. She stated that her vision was normal but that she wished advice concerning the vertical elongation of her left pupil. This phenomenon was first noticed by her dentist in June. At that time her eyes were examined, and glasses were prescribed, which were discarded within two weeks.

A thorough physical examination by her family physician revealed no pathologic condition except an absence of the knee jerk reflex. This, however, had been known for several years, as life insurance had been refused on that one count. Her physical examination had included Wassermann tests of the blood and spinal fluid, both of which were reported negative. She denied having had a serious illness or a recent injury. She was a very healthy-appearing person who weighed

1. Fuchs, E.:   Text-Book of Ophthalmology, ed. 8, Philadelphia, J. B. Lippincott Company, p. 668.

2. Barr, Albert S.:   Essential (Progressive) Atrophy of the Iris; Report of a Case, Arch. Ophth. **12**:567 (Oct.) 1934.

140 pounds (63.5 Kg.) and was physically active, engaging in such sports as swimming, tennis, horseback riding and, occasionally, golf. There was a history of an injury in 1920. The patient fell when racing on ice skates, striking her forehead and being knocked unconscious. She was told of a slight concussion, but recovery was complete in a few days, and it is difficult to believe that the accident so many years previously would have any connection with this ocular condition.

Ophthalmic examination showed vision of the right eye to be 20/20 + and that of the left 20/20 —. The right eye appeared normal. It was quiet; the iris was light bluish green, and the pupil was round and active. The left eye was quiet, and the color was the same as that of the right, but the pupil was not round. There was a vertical angle superiorly, as shown in *A* of the figure. The pupil had the shape of a hanging drop. The slit lamp study showed no evidence of inflammation. The fields and blindspot of each eye were plotted and were normal. Examination carried out with the pupils under the influence of a cycloplegic revealed practically no refractive error. The tension, recorded with a new Schiötz tonometer, was 20 mm. in each eye. The fundi were normal.

On November 20, or nearly three months later, the patient was seen again. Her pupil was greatly elongated vertically and pulled slightly temporalward (inset in the figure). The patient reported for observation every three or four months.

In February 1935 a drawing was made which depicted some real progressive atrophy of the iris (*B* of the figure). The fields were normal, and the tension was 15 mm. for the right eye and 20 mm. for the left. The slit lamp showed the stroma of the iris to be drawn upward and temporalward. Between 12 and 2 o'clock only the pigment layer of iris was left, and in some parts of the irregular pupillary area there was a uveal ectropion.

In November 1935 there had been more extensive atrophy and disappearance of stroma of the iris, the barren area extending from 12 to 2 o'clock and toward 3 o'clock. The tension and the fields had remained normal. There had also been a disappearance of the pupillary area of pigment layer in the upper temporal quadrant (*C* of the figure).

In February 1937, nearly four years after the onset of the disease, the first anterior synechiae were discovered by use of the corneal microscope. The pupillary area of the iris below and nasally seemed to be thinner and tense, and the iris was adherent to the corneal endothelium in an area below, about 2 mm. from the limbus and extending 4 mm. horizontally, and one adhesion was seen superiorly, toward 11 o'clock and about 2.5 mm. from the limbus (*D* of the figure). Dr. M. U. Troncoso made a gonioscopic examination at this time and reported as follows:

"The iris has a faded gray color, and its ridges look more distinct than in the normal eye. The nasal border of the coloboma shows distinctly the normal uveal rim, while in the temporal, and especially in the lower, part, the border of the iris has been split in its two layers. The retinal layer is in place and shows a peculiar meridional striation. The stroma layer is atrophic and has receded downward, leaving a wide, triangular area exposed. The examination of the angle of the anterior chamber at this place showed a wide anterior peripheral synechia (*E* of the figure), which is attached to the limbus rather forward, almost to the transparent edge of the cornea. This synechia is partial. On each side the angle is open, the brown band of the ciliary body being clearly observed. The canal of Schlemm is not apparent and merges with the white sclera. In the upper part of the angle the coloboma does not reach the scleral limbus. A narrow stump

of the retinal layer of the iris remains. Behind it the ciliary processes appear and show no evidence of inflammatory disease. On the nasal side of the coloboma there is also a narrow anterior peripheral synechia. Except for these synechiae the angle is open all around the limbus."

In none of the reported cases of this disease has gonioscopy been used and the angle of the anterior chamber studied during the life of the eye. The discovery of these peripheral synechiae may be important from the standpoint of pathogenesis. In a majority of the cases the synechiae were reported after histologic examination of the enucleated eye.

<center>COMMENT</center>

The first cases of this unusual disease were reported in Europe in the last decade of the nineteenth century. The first clearcut clinical description of this condition was written in 1903 by Harms.[3] In his group he included G. Lindsay Johnson's [4] case of progressive atrophy of the iris, reported in 1886, and Carl Hess' [5] case of "non-traumatic iridodialysis," the report of which appeared in 1892. The earliest case of this condition described in the American medical literature is that reported by Casey Wood [6] in 1908, who described his case in detail in 1910 at the meeting of the Section on Ophthalmology of the American Academy of Ophthalmology and Otolaryngology. Among the earlier cases were those reported by Zentmayer [7] in 1918, de Schweinitz [8] in 1915 and Lane [9] in 1917.

Fine and Barkan [10] in June 1933 saw a 9 year old patient who had had changes in the iris of each eye for over four years. These were noticed by the family before the child was 5 years old. The authors' descriptions of the early increase in the tension of each eye, of the ineffective operative treatment and of the thinning of the sclera, which became bluish, promptly places this condition in the class of buphthlmos. It was evidently congenital and progressive and will terminate as true buphthalmos always does. It does not earn a place among the true essential progressive atrophies of the iris.

The eccentric position of the pupil is the earliest symptom of essential progressive atrophy of the iris. This sign has been particularly stressed by illustrations in the reports of the case histories. The irregular enlargement of the pupil has always been described, and several authors

3. Harms, C.: Klin. Monatsbl. f. Augenh. **41**:522, 1903.
4. Johnson, G. Lindsay: Ophth. Rev. **5**:57, 1886.
5. Hess, Carl: Klin. Monatsbl. f. Augenh. **30**:103, 1892.
6. Wood, Casey: Ophthalmoscope **8**:858, 1910.
7. Zentmayer, William: Am. J. Ophth. **1**:510, 1918.
8. de Schweinitz, George E.: Tr. Am. Ophth. Soc. **14**:250, 1915.
9. Lane, L. A.: Ophth. Rec. **26**:285, 1917.
10. Fine, Max, and Barkan, Hans: Am. J. Ophth. **20**:277, 1937.

have offered explanations of these changes. Rochat suggested that the change in the size and position of the pupil may be "the result of a circumscribed shrinking of the iris by some pathological process" or "by assuming a primary atrophy of the iris, in which case the pupil would naturally be drawn to the side where the iris is less atrophic." This explanation may well be applied to the condition in my case. The pupil, which was first vertically elongated upward, was later displaced downward, being dragged by the less atrophic iris, before the progressive atrophy advanced into the upper temporal quadrant.
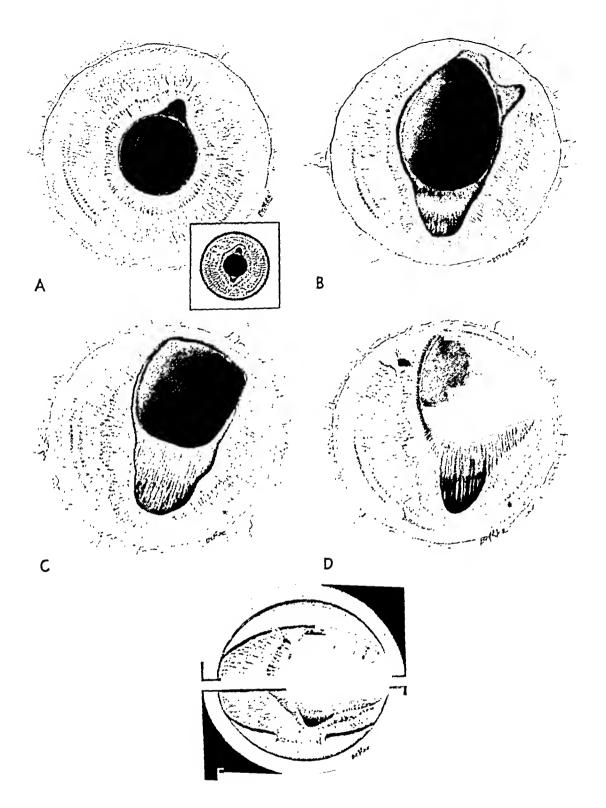
It is interesting to note in the study of the records of cases how few cases were studied before the onset of the increase in the tension and other symptoms of glaucoma. De Schweinitz' patient was under observation for two years before any sign of glaucoma appeared. Rochat and Mulder's [11] patient was seen in 1917, when the atrophy of the iris was well advanced and the eye was entirely free from any sign of glaucoma. These authors could not report the date of appearance of the signs of glaucoma, as the patient was not seen for six years. When the patient did report, the eye was blind and in a state of absolute glaucoma. My patient has been seen fairly regularly, and accurate observations have been made. Her vision is 20/20, and her fields and fundus are normal. The fact that her age was 29 at the onset makes the condition fit very well in de Schweinitz' classification as to age and sex. He listed seven females and five males, and the ages of the females ranged from 20 to 47.

Although it has been suggested that some constitutional fault might be an etiologic factor in this unusual disease, each case report has stated that the general physical examination has usually resulted in negative findings.

In all the reported cases the disease has been unilateral. Feingold [12] expressed the belief that in his case there may have been congenital vascular disturbances near the lesser vascular circle, which could have induced changes in the iris. Lane stated the opinion that a disturbance of circulation due to sclerosis of the vessels of the iris might be a causal factor. Lane's patient had latent tuberculosis. Wood's patient was reported to have had fibrinoplastic iridocyclitis, and it is possible that the condition was not true essential atrophy, as low grade uveitis is not accepted as a causal factor. De Schweinitz reported that his patient had had an attack of retrobulbar neuritis of the opposite eye two years previously and that "although it might be the forerunner of a disseminated sclerosis, it could hardly be related to the iris lesions." Zentmayer suggested that sclerosis of the vessels of the iris cause loss

11. Rochat, G. F., and Mulder, W.: Brit. J. Ophth. 8:362, 1924.

12. Feingold, M.: Am. J. Ophth. 1:1, 1918.

Appearance of the eye. *A* shows the appearance in September 1933, and the inset shows the appearance on November 20. *B* shows the condition as seen on Feb. 19, 1935; *C*, the appearance on Nov. 19, 1935; *D*, that on March 4, 1937, and *E*, the gonioscopic view on March 4, 1937.

of nutrition, which might create atrophy of the stroma, but he did not advance a theory for the cause of this vascular sclerosis. In de Schweinitz' first paper in 1915 it was stated that "a toxin liberated by some constitutional or local infection may be responsible for exciting iris-vascular disease, whereby nutrition is lowered and atrophy follows."

The treatment of the disease has been very unsatisfactory. In a majority of the cases in which treatment with miotics or surgical intervention has apparently been successful, the results have been only temporary. One reluctantly, yet assuredly, accepts the prognosis that in the long run the eye will be lost because of absolute glaucoma. This thought, however, is somewhat overshadowed by the fact that the disease has never affected both eyes of the same patient.

extensive corneal scarring. When she returned to our care the lids had been closed for seven months.

Direct observation verified her impression that the eye was dry, but it was obvious that some secretion of tears was present. The adhesion of the lids was divided so that more complete examination could be carried out. There was extensive central scarring of the cornea in the anterior layers; the conjunctival vessels were injected, especially on the nasal side, and vision was 6/30 with correction. Measurements proved that secretion of tears was present on the involved side, but it was only one sixth of the amount in the left eye.

Both canaliculi of the right eye were closed by electrocoagulation. They remained blocked for two days, during which time the eye was wet. On the third day the eye appeared dry again, and the canaliculi were proved to be open by syringing. With the drying that followed, pitting of the epithelium was first noticed. The lower canaliculus was then slit by the actual cautery, and it became permanently closed. The eye was more moist but not as much so as it had been immediately following electrocoagulation of both canaliculi. Therefore, the upper canaliculus was subsequently treated with the actual cautery. Though this did not close the canaliculus permanently, it produced ectropion of the punctum which effectively blocked drainage. Since then the eye has been wet but not too troublesome, and the pitting of the epithelium has disappeared, as has the congestion of the conjunctival vessels. The subjective sensation of discomfort, which was present even when the lids were sutured and during the time when the eye was dry, is no longer noticed. The patient was aware of an improvement in vision, which is now equal to 6/15 with correction.

COMMENT

The satisfactory result following closure of the canaliculi has been maintained over a period of five months. It is comparable to the results described by Beetham[3] after he had closed the ducts in patients with filamentary keratitis.

Although closing the canaliculi would seem to be a simple procedure, owing to the fact that sensation is lost and no anesthetic is required, the canals tend to become patent again. When the canaliculus was slit with the actual cautery it closed permanently, and therefore such a procedure would seem to be the one of choice.

The incidence of neuroparalytic keratitis after complete section of the posterior root of the fifth cranial nerve or destruction of the ganglion by alcohol is difficult to determine, as it may occur many years after the therapeutic surgical intervention. Wilfred Harris[4] expressed the belief that it probably occurs in about 20 per cent of the cases. Other authors have stated the belief that 10 per cent is a fair estimate, but both these percentages seem high to us.

If the important factor in the development of the corneal lesions is diminished secretion, it should be possible by detailed physiologic tests to select the cases in which this complication is likely to develop and to prevent it.

3. Beetham, W. P.: Filamentary Keratitis, Tr. Am. Ophth. Soc. **33**:413, 1935.
4. Harris, Wilfred: The Facial Neuralgias, London, Oxford University Press, 1937, p. 62.

We have studied the lacrimal and parotid secretion in every patient who has been operated on for trigeminal neuralgia in the Montreal Neurological Institute in the past year. Measurement of the secretion of tears has been carried out by placing twisted absorbent cotton pledgets in the internal canthus of each eye so that they cover both puncta. These pledgets are allowed to remain in situ for five minutes. The difference in weight in milligrams before and after insertion is taken as an index of the amount of secretion. To simplify the procedure and make it as accurate as possible the dry cotton pledgets are placed in glass weighing jars with ground glass stoppers and weighed. The pledgets are then removed with a forceps from the jars and placed in the eyes, then returned to their respective jars and covered with the ground glass stoppers to prevent evaporation and weighed immediately. We have used the Sartorius analysis suppression scales, which weigh to 0.1 mg., and they have always been operated by the same technician. For the measurement of parotid secretion the same technic has been used, except that nasal tampons have been placed over Stensen's ducts, as suggested by Poth.[5]

When repeated tests have been carried out on a patient the results, as might be expected, have varied. We have obtained, however, definite information as to the presence of secretion and a fairly accurate impression of its amount. We have felt that the weighing method when done properly is more valuable than Schirmer's[6] test with filter paper. It has been surprising to find that the secretion from the parotid gland is usually reduced on the affected side when the lacrimal secretion shows reduction. Since this measurement is a coarser procedure and causes less discomfort to the patient it may eventually prove to be the more valuable test, but more data are required before this conclusion can be drawn.

The problem of the secretory nerve supply to the lacrimal gland need not be considered here. It is as complex as that of the secretory nerve supply to the parotid gland, and our studies on patients have not served to clarify it. For example, Verhoeff's hypothesis that diminished secretion of tears is due to injury of the greater superficial petrosal nerve is contradicted by the findings in the case we have reported. In this case neither the greater superficial petrosal nerve nor the facial nerve was injured; yet there was diminished lacrimation, and keratitis developed. If the sensory loss were the all-important cause, the lesions should appear as a complication much more frequently than they do. We can see no reason for accepting a so-called trophic influence, as

5. Poth, E. J.: A Simplified Technique for Quantitative Collection of Salivary Secretions of Man, Proc. Soc. Exper. Biol. & Med. **30**:977, 1933.

6. Schirmer, O.: Studien zur Physiologie und Pathologie der Tränenabsonderung und Tränenabfuhr, Arch. f. Ophth. **56**:197, 1903.

first suggested by Magendie [7] and more recently advanced by Tagawa.[8] This complex theory has no adequate proof, in our opinion.

Another example of keratitis due to drying is keratitis e lagophthalmo. It is seen commonly in the comatose patient who lies with the eyes open or only partially closed, with no blinking and no conscious discomfort. Corneal lesions seldom develop after paralysis of the seventh nerve. Although the patient does not wink, the discomfort which drying causes stimulates him to roll the eye up under the lid and thus moisten the cornea. The drying of the cornea occurs chiefly during sleep, but even then the eye tends to roll up so that the cornea is partially covered. The lack of winking and ectropion of the lower punctum present after palsies of the seventh nerve, with the consequent damming back of tears, must also be a beneficial factor.

Years ago Magendie observed in animals that the eye in which keratitis developed following section of the fifth nerve was dry. He dismissed drying as the cause of the lesions, because when he took out the lacrimal gland in other animals corneal lesions did not develop. It is now recognized that corneal lesions do not develop after excision of the lacrimal gland because the secretion of the accessory lacrimal glands, such as Krause's glands, is usually adequate. In keratomalacia, though the eye appears to be dry this is not really the case.

The occurrence of keratitis after total rhizotomy for the relief of trigeminal neuralgia is such a serious complication that the following routine is suggested: Oil should be dropped in the eye immediately after operation and a Buller's shield applied. The shield should remain in place until it can be determined that the eye is not dry. If the secretion of tears is adequate to prevent injection of the conjunctival vessels when the eye is left uncovered and every precaution is taken to avoid trauma, no complications are to be anticipated. The patient is discharged from the hospital with Adson and Schroeder's [9] protective shield attached to spectacles. On the other hand, if tears are greatly diminished and the conjunctiva becomes injected, the canaliculi should be closed. This procedure causes no deformity and no special inconvenience. The conjunctival injection should be considered as an elective indication. Corneal pitting or loss of epithelium makes closure of the canaliculi an operation of necessity. Our experience with one case would suggest this to be adequate. Should it fail, tarsorrhaphy is imperative.

7. Magendie, M.: De l'influence de la cinquième paire de nerfs sur la nutrition et les fonctions de l'oeil, J. de physiol. expér. **4**:176, 1824.

8. Tagawa, S.: Ueber die Beschaffenheit der Hornhautoberfläche nach Trigeminusdurchschneidung, Arch. f. Augenh. **102**:231, 1929.

9. Adson, A. W., and Schroeder, A. A.: A Corneal Shield, J. A. M. A. **86**: 1835 (June 12) 1926.

ORBITAL CYST WITHOUT EPITHELIAL LINING

REPORT OF TWO CASES OF BLOOD CYST

JOHN M. WHEELER, M.D.

NEW YORK

Cysts without lining of epithelium can be found outside the orbital tissues. Of these, perhaps the best known are hydatid cysts of the liver, blood cysts of the breast, subdural blood tumors of the cranial cavity and, within the eyeball, cysts of the retina and serous cysts of the iris. A few types of cyst without epithelial lining within the orbit and outside the globe have been studied, and several reports of such cysts have been published. Examples of these are serous or exudation cyst, parasitic cyst, congenital cyst of the orbit associated with microphthalmos or with coloboma, cyst in the sheath of the optic nerve [1] and blood cyst. A serous cyst of the orbit may have its origin in the bursa between the tendon of the superior oblique muscle and the trochlea or in the bursa between the levator muscle of the upper eyelid and the superior rectus muscle. Cyst caused by parasites in the orbit must be rare in this country, although a number have been reported in Europe and Asia. A number of cases of congenital cyst associated with microphthalmos and anophthalmos have been reported. A congenital defect in the wall of the eyeball makes the development possible, and, starting in the lower part of the globe, the cyst may grow until it occupies a large part of the orbital cavity. A good example of cyst associated with coloboma is that reported by Calhoun.[2] Several cases of blood cyst in patients operated on for unilateral exophthalmos have been described. I should like to report two cases of this type of cyst.

REPORT OF CASES

CASE 1.—F. R., a 29 year old man, was referred to me by Dr. A. C. Snell because of unilateral exophthalmos. I first saw him on Oct. 11, 1934. About two years before this the right eye had started to be prominent. He gave no history of trauma or inflammation. The bulging of the eyeball had gradually progressed.

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1. Bane, W. C.: Cyst of Dural Sheath of Optic Nerve, Am. J. Ophth. **1**:17, 1918.

2. Calhoun, F. P.: · Bilateral Coloboma of the Optic Nerve, Associated with Holes in the Disk and a Cyst of the Optic Sheath, Arch. Ophth. **3**:71 (Jan.) 1930.

He had been under treatment for inflammation of the right frontal sinus and the antrum. Vision was normal in each eye. The right eye was displaced forward, downward and slightly outward. There was an exophthalmos of about 8 mm. The protruding eyeball showed no pulsation, and auscultation of the skull gave negative results. The patient said that the right eye was irritated and he slept with it half open, that there was watering of this eye, and that he had "pressure headaches." A plotting for diplopia was made, and it was found that the right eye had definite limitation of motility except in turning of the eyes to the right. The basal metabolic rate was normal. The interior of each eye was without abnormality. Roentgen examination by Dr. Pfeiffer showed well defined asymmetry of the orbits, with a pathologic state of the right orbit. The report was as follows:

"In the roof of the right orbit a spherical cavity measuring approximately 35 mm. lies adjacent to the large frontal sinus. The walls of this cavity are thin and are complete on all sides save below, where the cavity encroaches on the orbit. The content of the cavity is of diminished density and impresses me as being nearly clear. The roof of the cavity bulges upward 5 or 6 mm. The right optic canal is circular in contour and measures 5 mm. in diameter. The left optic canal is similar. The ethmoid cells adjacent to the right canal are clouded. The large cavity in the roof of the right orbit is typically that of a retention cyst."

With the patient under anesthesia induced by tribromethanol in amylene hydrate and ether I operated on October 13. Two intermarginal sutures were introduced to hold the margins of the lid in apposition during and for a few days following operation. An incision was made through the skin just below the eyebrow, and a large fluctuant mass was felt in the upper portion of the orbit just behind the orbital margin. The anterior part of the sac was exposed, and an incision was made in it. A large quantity of greenish fluid about the consistency of pus was evacuated. A part of the wall of the cyst was removed for microscopic study. After the fluid contents of the cyst were removed, a thorough application of pure phenol was made to the interior of the wall of the sac, and it was well neutralized by alcohol. The deep structures were brought together by chromic gut sutures, and the incision in the skin was closed with interrupted silk sutures. A pressure dressing was applied in order to hold the collapsed walls of the cyst in apposition, and the pressure was maintained for a week. Recovery was uninterrupted, but there was numbness up to the hair line on the right side of the forehead.

The pertinent part of Dr. Reese's report on the microscopic examination is as follows:

"The specimen consists of dense fibrous tissue, the older being external and that of more recent production mostly internal, with gradations between. Throughout the inner half of this fibrous tissue are many lymphocytes, in some places forming focal accumulations. The internal surface shows no epithelial lining, but instead there are some fragments of fibrin. Over a large portion of this wall plaques of bone have been laid down. There is no evidence of tuberculosis. There is no evidence of malignancy. The impression is that this could well be the result of the organization of a large hemorrhage."

Samples from the contents of the cyst showed no bacteria with either gram stains or acid-fast stains.
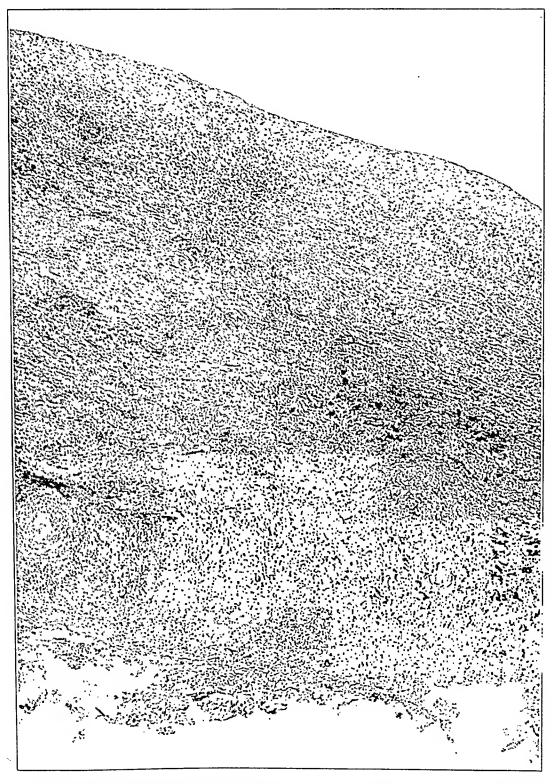
This is almost surely an example of blood cyst of the orbit, although there was no history of traumatism. Probably there had been no recent hemorrhage, as the fluid was green rather than red.

CASE 2.—On Dec. 24, 1936, Dr. Charles A. Elsberg referred to me J. H., a man 45 years of age. In April 1935 during a routine physical examination Dr.

Theodore Sanders noticed bulging of the right eye. The patient was referred to an ophthalmologist, who thought the patient should be in the hands of a surgeon specializing in work on the brain, and he was sent to Dr. Elsberg, who kept him under observation until he was referred to me. During the early months of proptosis a tentative diagnosis of lipoma of the orbit had been made. Roentgen therapy had been administered, without benefit. A few days before I saw the patient, while playing with his son he had experienced the feeling that the right eye was "popping out" and the upper lid caught back of the eyeball. On December 24 vision of the right eye was 20/20 — 4 and that of the left eye 20/15 — 2. The patient accepted a + 1.00 D. sph. ⊃ + 0.25 D. cyl., ax. 55 for the right eye, with which vision was 20/20, and a + 0.25 D. sph. for the left eye, with which vision was still 20/15 — 2. There was exotropia of 20 degrees for distance and 25 degrees for near vision. A plotting for diplopia showed exotropia in all directions of gaze. The separation of the images was greatest when the eyes turned to the left and upward, but the imbalance was not far from comitant. There was exophthalmos of the right eye of 6 mm. The palpebral fissure was longer and wider in the right eye than in the left. The measurements of the width of the fissures were as follows: with the eyes looking straight ahead, 14 mm. for the right eye and 9.5 for the left eye; with the eyes looking up, 17 mm. for the right eye and 11.5 mm. for the left eye, and with the eyes looking down, 6 mm. for the right eye and 4.5 mm. for the left eye. The lower lid did not adhere closely to the globe for about 7 mm. from the outer canthus. The conjunctival vessels appeared dilated behind the equator, but there was no discoloration of the conjunctiva. Between the globe and the lateral orbital wall was a smooth, nonlobulated round mass which protruded slightly beyond the orbital rim; this protrusion held the outer part of the lower lid slightly away from the eyeball. Protrusion of the mass was increased by pressing the eyeball back into the orbit. There were no pulsation and no subjective or objective bruit. Fluctuation was easily elicited. The blindspots and the outlines of the fields were normal. The interior of each eye was normal. Dr. Dyke as a result of roentgen examination at the Neurological Institute made a tentative diagnosis of cyst of the right lacrimal gland. My diagnosis was orbital cyst below the lacrimal gland.

Dr. Pfeiffer gave the following report on the results of roentgen examination:

"Films of the orbits show the margins to be asymmetrical, the left to be normal and the right to be larger and to have a defect in the frontal process of the zygoma. This defect is concave, measures approximately 11 mm. and extends into the bone for 4 or 5 mm. and, seemingly, posteriorly for a short distance. The temporal line is normal. The margins of this defect, or area of diminished density, are irregular and may not be palpable. The right orbit seems slightly larger and shows increased density of soft tissue. The optic canals appear to be circular and symmetrical and measure approximately 5 mm. in diameter. All the paranasal sinuses are large and clear. The sella turcica measures 9 by 12 mm. and is not eroded."

On December 29 I operated on this patient while he was under anesthesia induced by tribromethanol in amylene hydrate and ether. A horizontal incision was made from the outer canthus backward over the zygomatic bone. The tarso-orbital fascia was freely incised along the orbital margin, both above and below the horizontal incision, and the conjunctiva was separated from the globe on the temporal side. A smooth-surfaced, brownish, fluctuant mass could be seen and felt. It was freed on the medial surface by blunt dissection with the finger and the curved scissors. It was not attached to the lateral rectus muscle but to the outer wall of the orbit from the orbital margin back about 30 mm. It rested on the floor

Photomicrograph of the wall of the orbital cyst in case 2. There is absolutely no lining of epithelium or endothelium.

of the orbit but was not adherent to it. By mistake the wall of the cyst was ruptured, and a quantity of red fluid escaped. The fluid was only a little darker than fresh arterial blood. The unattached portion of the wall of the cyst was dissected out and sent to the laboratory for examination. Palpation of the lateral wall of the orbit revealed a depression approximately 6 mm. in diameter, with a rim of firm tissue along the upper border of the depression. The part of the cyst attached to the outer wall of the orbit was sponged dry and treated with pure phenol; this was followed by washing with alcohol. The septum orbitale was closed with catgut sutures, and the incision in the skin was closed with fine silk sutures. Before the dissection was started, two silk sutures had been placed to hold the margins of the lids together. These were allowed to remain, and a pressure dressing was applied. Healing was uninterrupted.

Dr. Reese gave the following report on the microscopic examination of the wall of the cyst:

"The specimen is the wall of a cyst in which *no epithelial elements are noted* (the appearance is shown in the figure). The tissue is merely a fibrous wall, the inner layers of which are edematous. Throughout all the layers there is an infiltration of lymphocytes and a few polymorphonuclear leukocytes. Particularly noticeable is a broad and definite layer of pigment-bearing cells which occurs just beneath the internal surface of the wall. The pigment has the appearance of being of a hematogenous nature and has resulted from the phagocytosis of hemosiderin crystals from old blood. There are some cholesterol crystals in the wall of the cyst, and a few are surrounded by foreign body giant cells. There are also pus cells, lymphocytes, various larger unrecognized cells which are probably edematous fibroblasts, and other types of cells. There is no caseation. Sections stained by Perl's method show hematogenous pigment, and those stained by the Fontana method show no melanin."

Dr. Purdy Stout studied the slides and reported as follows:

"The lining of the cyst is made up of fibrous tissue in which there are many phagocytes full of blood pigment, foam cells loaded with lipoid, and many capillaries in isolated areas. There seems to be some degeneration but scarcely any evidence of inflammation. This is the picture which one sees in cyst that develops from areas of traumatic necrosis of fat. My associates and I have seen this type of cyst in the breast in persons from whom no history of trauma could be obtained. I cannot offer any other explanation for this cyst."

Dr. Karl Meyer examined the fluid content of the cyst and reported that it was of red blood color and contained innumerable intact red blood cells. There were also abundant typical plates of cholesterol. On dilution with saline solution a thick film of cholesterol crystals floated on the surface and adhered to the sides of the container. The quantity of cholesterol was found to be 15 per cent of the total dry weight.

On March 9, 1937, a little over two months after operation, the exophthalmometer readings were 22 mm. for each eye. The diplopia had changed little.

COMMENT

In case 2 probably there had been a fresh hemorrhage into the cyst a few days before I saw the patient, when the eye had a "popping out" feeling, and the fresh hemorrhage accounted for the blood red color of the fluid contents. In case 1 probably there had been no bleeding into the cyst for a relatively long time before operation, and so the contents were not red but green.

In neither of these cases was a significant history of traumatism elicited, and it may be that traumatism is not necessary for the development of a blood cyst within the orbit. A blood tumor in the breast may appear in an area involved in traumatic necrosis of fat, or it may appear in a patient without a history of trauma. A persistent subdural hematoma may occur without direct traumatism to the area involved.[3]

In 1932 Crigler [4] reported a subchoroidal hemorrhage in the left eye of a woman 44 years of age who fell to the floor, landing on her right knee, with only an indirect jar to the head.

Against this there may be a definite history of orbital injury, as in the case of Denig's [5] patient, who received an orbital injury while fighting a duel.

Awerbach [6] reported three cases of hemorrhagic tumor of the orbit, in two of which there was a definite history of· injury years before he examined the patients.

Michaïl [7] reported a blood cyst that resulted from the presence of a wad of cotton sponge that was left in an orbit at the time of an enucleation. He operated on the blood cyst ten years after the enucleation.

Vasomotor disturbances (such as those occurring in association with the menopause), hemophilia and alteration in the endothelium of the orbital blood vessels have been mentioned in connection with the etiology of hemorrhage into the orbit.

In a discussion of blood cyst of the orbit attention might be called to the fact that there may be bleeding into an epithelium-lined cyst, giving the contents a red color which is evident when the wall of the cyst is opened. Authors have called attention to defects in the epithelial lining of walls of a dermoid cyst. At the meeting of the American Ophthalmological Society in 1936 Bernard Samuels [8] reported that among thirteen dermoid cysts he found defects in the epithelium in the walls of three. The epithelial lining was broken by patches of granulation tissue, and it is likely that hemorrhage may occur in such areas from new-formed vessels.

3. King, Clarence: Chronic Traumatic Subdural Hematoma as a Cause of Choked Disc, Am. J. Ophth. **20**:149, 1937.

4. Crigler, L. W.: Subchoroidal Hemorrhage Diagnosed as Sarcoma of the Choroid, Arch. Ophth. **8**:690 (Nov.) 1932.

5. Denig, Rudolph: Subperiostal Bloodcyst of the Orbit: Report of a Case, Ophth. Rec. **11**:187, 1902.

6. Awerbach, M. I.: Les tumeurs sanguines de la cavité orbitaire, Ann. d'ocul. **170**:863, 1933.

7. Michaïl, D.: Cyste hématique de l'orbite, Arch. d'opht. **52**:851, 1935.

8. Samuels, Bernard: Dermoid Cysts of the Orbit, Arch. Ophth. **16**:776 (Nov.) 1936.

In most cases of hemorrhage into the orbit absorption of blood occurs, and it is not known why occasionally a wall of connective tissue forms around the blood. Neither is it known why blood within the sac may be found unclotted.

Osmosis may be a factor in the growth of a blood cyst in the orbit, but probably in most cases growth comes through recurrent hemorrhages. Probably the wall is formed as a result of reaction of the orbital tissue to the presence of material foreign to it.

Removal of the entire wall of the cyst is not necessary, whether there is any epithelial lining or not. Generous application of phenol will destroy epithelium, endothelium or the surface of connective tissue. Then if heavy pressure is applied over the lids (while they are held together by intermarginal sutures) there will be apposition of the walls of the cavity and healing between them.

In operating the surgeon can get a good exposure of the orbital contents without an osteoplastic flap, as in the Krönlein procedure. Free canthotomy, with continuation of a horizontal incision beyond the orbital margin and with free incisions of the tarso-orbital fascia along the orbital margin above and below, will allow of good retraction of flaps, with exposure of the wall of the cyst.

DISCUSSION

DR. ALGERNON B. REESE: Dr. Wheeler has described an unusual type of orbital cyst. In passing he has mentioned another rare type of cyst of the orbit which also has no epithelial lining, that is, the congenital cyst associated with coloboma, or retinocele. This usually is found in association with microphthalmos, and then the diagnosis is obvious. Occasionally it is present in an eye of normal size and appearance, and then the diagnosis may be difficult. Two microscopic specimens of such a cyst were shown. The cyst may be larger than the eyeball. The wall is composed of tissue resembling that of the sheaths of the optic nerve and is lined by neuroglial tissue and sometimes by retinal elements. The cyst produces exophthalmos which is noticed usually at birth but which increases after birth so that it may lead to an ulcer e lagophthalmos. Determination of the condition of the optic nerve usually leads to the correct diagnosis. The nerve head may show the congenital remains of tissue over its surface, or there may be a colobomatous cupping. The question of an orbital growth or inflammation as a possible etiology may arise.

LATE RESULTS OF EXTRACTION OF CATARACT

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The late results of extraction of cataract cannot be known from the statistics of great operative clinics, whether situated in India, Vienna, London or America. To such clinics patients go from long distances, to remain until the operative wound may heal and sometimes until a first trial of vision with glasses can be recorded. Rarely does the patient return to such a clinic to report the ultimate, or late, results of his treatment. The permanence of the resulting vision and its total value to the patient can be more certainly judged by the results in patients seen in private practice and followed to the end of life or at least for some years after the operation.

REVIEW OF CASES

A diabetic patient with double cataract went to Vienna, where one lens was extracted. The next year he went to Wiesbaden, where the other lens was extracted. He came to me eighteen months later to see if his vision could still be improved. He showed successful operative results in each eye. The extractions had been done with iridectomy, the capsule being left. He wore glasses that had been prescribed a few weeks after the last operation—spherical lenses, giving vision of 0.3 in each eye. Lenses were prescribed fully correcting the astigmatism. These gave vision of 1.3 in each eye, and this vision continued until his death, three years after his last operation.

In the other patient with diabetes included in this series the condition was mild, and there were no retinal lesions or much general disturbance. The patient lived three years after the extraction; vision decreased gradually from 0.5 to 0.25, and she gave up reading. Years ago a patient was seen in consultation who died in diabetic coma a week after extraction of cataract. The condition was severe, but the patient wished to see again before she died. That was before insulin was available to control the condition for operation and even before dietetic treatment had been brought to its present efficiency.

A woman of 65 had been blind in the right eye from cataract. She had it operated on, but an opaque capsule, not effectively divided, left the vision still poor. In another city she had extraction of cataract done on the left eye, and one month later had vision of 20/100 in this eye. But vision in this eye progressively failed. Five months later she came to me, with vision of perception of movements of the hand and chronic uveitis that had left vitreous opacities. Five years later her right eye has vision of 0.25 with her correcting glass. But the left eye is hopelessly damaged, probably from use too soon after operation. There was no other evident cause for the uveitis.

Neither of these extractions was done by me. But the results were evident, and such late results must be considered in forming a just

estimate of the value of extraction of senile cataract. When living in Philadelphia I observed cases of slow failure of vision after extraction of cataract done by surgeons of the highest skill. A few such failures are due to atrophy of the optic nerve or disease of the central nervous system. But the results in the majority of cases impressed the lesson that ample time, often several months, must be allowed the eye for recovery after operative treatment that so disturbs its whole nutritive mechanism. In a few cases in which both eyes had been subjected to extraction of cataract it was the eye that required the longer time for recovery that showed the better result.

*Table of Cases**

| | Age of Patient, Years | | | Vision | |
Case	At Operation	When Seen	Complications	Before Extraction	After Extraction
1	53	68	Myopia	Perception of light	0.80
2	37	75	Myopia; uveitis; corneal scar	Perception of light	0.50
3	61	63		Perception of light	1.10
4	48	49	(Traumatic cataract)	Perception of light	0.40
5	65	72	Uveitis	Perception of light	0.25
6	66	69	Myopia	Perception of light	0.90
7	75	77	Myopia	Perception of light	0.80
8	42	48	Diabetes	Perception of light	1.20
9	68	70	Asthma	Perception of light	0.70
10	60	62		0.03	0.80
11	52	65		0.10	1.00
12	63	75		Perception of light	0.60
13	66	68	Glaucoma	Perception of light	0.80
14	54	59	Corneal scar	0.06	0.50
15	32	34	(Traumatic cataract)	Perception of light	0.30
16	36	41	Tetany	0.03 0.20	1.00 1.10
17	57	71		0.04	0.20
18	54	70	(Traumatic cataract)	Perception of light Perception of light	1.20 1.20
19	32	43	Tetany	0.20 0.30	1.10 1.10
20	64	67		Perception of light	1.00
21	35	62	Uveitis and glaucoma	Perception of light	1.20
22	57	77	Myopia	Perception of light Perception of light	1.00 1.20
23	70	91	(Hypermature cataract); glaucoma	Perception of light	0.50
24	72	75	Diabetes	0.10	0.50
25	60	68	Myopia; orbital cancer	0.05	0.80
26	70	72		0.07	0.60
27	77	80		Perception of light	0.45
28	71	73		Perception of light	0.45
29	73	78		Perception of light	0.80
30	74	84		Perception of light Perception of light	1.00 0.50
31	65	70		0.10 0.10	0.90 1.00
32	74	76		Perception of light	0.50
33	64	68	Myopia	Perception of light	0.80
34	66	71		Perception of light	0.80
35	61	72		Perception of light	1.10
36	55	61		0.10	1.10
37	60	62	Myopia	0.15	0.80
38	50	52	Myopia	0.01	0.40
39	60	67	Tobacco amblyopia	Perception of light	1.00
40	78	80	Loop extraction	0.03	0.20
41	68	70	Loop extraction	Perception of light	0.10
42	83	93		Perception of light	0.12
43	72	77		Perception of light	0.60
44	43	48		Perception of light	0.60
45	75	77		Perception of light	1.00
46	70	72		0.10	0.40
47	73	75		Perception of light	0.90
48	56	65	Glaucoma	Perception of light	0.10
49	54	64	Myopia; uveitis	Perception of light	0.60

* Resulting vision was 1 or more in nineteen eyes.

Among the cases that furnish the chief basis of this paper were four in which cataract was complicated by glaucoma.

In one of these cases a woman of 56 suffered from facial erysipelas, and while she was recovering from this bilateral acute glaucoma developed. Iridectomy for glaucoma was done on the left eye, about one fifth of the iris being removed. The right eye seemed to recover under the use of physostigmine. About a year later the patient was taken to a prominent general surgeon, who had also been one of the surgeons of the Wills Hospital, to see if the right eye should also be operated on. The surgeon wrote to the patient's family physician that there was no sign of glaucoma in either eye and, except for the iridectomy wound, there was no sign that she ever had glaucoma, but that she was getting cataract. Cortical striae were noted in both lenses at the time of the iridectomy. Ten years after that I was called to see her for the cataracts. There was absolute glaucoma in the right eye, but the left eye had good perception of light. Extraction of a mature gray cataract gave her vision of 0.1, which she had retained two years afterward.

Of the other patients with glaucoma, one had glaucoma before the extraction, and the condition was probably chronic; vision was little improved by operation. Two patients had moderately increased tension following needling operations for capsular opacity, which quickly subsided under the influence of miotics. One patient had a sharp rise of tension after preliminary needling of the lens. Extraction was done a few hours later. The patient has had no evidence of glaucomatous attacks in the twenty-seven years that have followed.

For the four cataracts associated with tetany, treatment with calcium and parathyroid preparations and thyroid extract was tried for some months, without any decidedly beneficial effect. Extraction of the cataracts brought restoration of standard vision in all four eyes, which continued for the two to five years that the patients remained under observation.

Myopia has been included among the causes of cataract. The findings in these cases give some support to that idea. But the proportion of previously myopic eyes in the patients, eleven in fifty-five, is not much greater than the norm of 18 per cent for all eyes after the age of 50 years.[1] The late results in these cases seem to indicate that such eyes bear extraction of cataract as well as most other eyes. In case 49 both eyes had been affected by chronic uveitis, and the fellow eye had become entirely blind, and had been enucleated. But since extraction of the cataract vision in the eye has improved until now it is 0.6, and the patient can do her own reading, which was previously impossible.

In case 2 there had long been uveitis with myopia. The eye on which extraction was done had been the worse eye and had not been used for reading, until the other eye had the macula damaged by a choroidal lesion. Extraction of the lens has given it vision of 0.5, and the patient uses her eyes more freely than she has for many years.

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1. Jackson, E.: Norms of Refraction, Tr. Sect. Ophth., A. M. A., 1931, p. 179.

Eyes blind from uveitis often show cataract eventually. In case 4 one eye had been lost in this way after the extraction. But the other eye has vision of 0.3, and is still improving after seven years. The important point is that most causes of uveitis can be removed entirely; and if this is done long enough before the operation the history of previous uveitis is no bar to the extraction of cataract. The patient in case 21 had uveitis, for which he was treated at the Hospital of the University of Pennsylvania, ten years before he had cataract. There was no recurrence of the uveitis at any time. Twenty-seven years after the removal of the cataract vision in that eye is as good as that in the fellow eye, which has never been diseased. Furthermore, with the full correction for each eye, differing about 9 D. for the two, the patient has little difficulty when he uses both eyes together.

It should be noted that in all the cases the operation was done with opening of the capsule in my usual manner, and in only a few was there need of later division of the capsule. It was interesting to note in Colonel Wright's lectures that at Madras, India, where the result had to be judged shortly after extraction, capsulotomy was judged the safer operation. In no case in this series was there any detachment of the retina or of the choroid. I have seen these complications in a few cases of extraction of cataract done by other surgeons, but they have never followed my extractions of cataract, except in two cases, in which the eye was destroyed by expulsive hemorrhage. In three cases of this series the operation was done with the patient sitting up in a high-backed chair, in which he was kept for many hours, to prevent expulsive hemorrhage, which there was reason to fear. Twice I have seen such hemorrhage checked by raising the body to the erect position and letting the feet drop at the side of the operating table.

TECHNIC OF OPERATION

The methods and technic of operation have been held to determine the results of extraction of cataract. This is largely true for the immediate, or early, results, and they may also be responsible for the late results. They may properly be considered with reference to this series of cases. In case 2 the operation was done by Dr. Allen Greenwood; in cases 19 and 49 both extractions were done by Dr. Wilmer. In the other cases extraction was done by me, and certain features of the operations may be properly considered to have influenced the results.

The cornea has more rigidity than the sclera. The cornea deserves a name that allies its physical properties with those of horn. The sclera is simply a very thick, tough but flexible membrane. When the corneal incision is made to extend at both ends into the sclera the advantage of the corneal rigidity is lost, and when pressure is applied to the eye the

corneal flap slides up on the part attached to the sclera. If the incision is made at the limbus, with a Graefe knife, it tends in the middle, which is cut last, to drag the sclera out of shape and thus disturb the relations of its contents, the ciliary body, choroid, retina and vitreous. And when the diaphragm between the aqueous and the vitreous is destroyed by intracapsular extraction the effect of this disturbance is increased and may be made permanent. This probably is the cause of the hammock, or updrawn, pupil.

Recognizing that the Graefe knife is excellent for fixing the puncture and counterpuncture but poor for completing the incision, I early designed a modified knife for extraction of cataract. This was first used on the patient in case 48 in 1887, and its improved form was described in the Transactions of the American Ophthalmological Society in 1890.[2] This knife was used in all the cases of this series except three. The incision was made with the puncture and the counterpuncture exactly in the limbus. Then, when it seemed desirable to make a conjunctival flap, the edge was turned a little toward the sclera as the incision was completed, and brought out beneath the conjunctiva. This produced a rather narrow flap but one that seemed to serve as efficiently as a broader flap.

The opening of the capsule of the lens was done with the cataract knife, not while making the corneal incision, but afterward. The knife was dipped in boiling water to cleanse it, and the back was introduced into the corneal incision and drawn back until the point was at the temporal edge of the pupil. Then the point was made to penetrate the capsule of the lens and pushed forward, making an incision in the capsule parallel to the corneal incision. This incision always permitted easy expulsion of the lens, and this procedure was rarely followed by need of a secondary operation.

Corneal or conjunctival sutures were not used for closure of the wound; nor was a tendon suture used for fixation. Fixation was made complete by grasping the conjunctiva with the fixation forceps just below the point for the counterpuncture. The impression grew on me that in doing this operation the anterior capsule always retracted and never gave trouble. Only when there was hemorrhage into the anterior chamber or an inflammatory exudate was subsequent needling required.

The nutrition of the eye is in several respects highly specialized and should be disturbed as little as possible by any operative procedure. Constant respect for the limitations this imposes seems to me the most important condition for operations for cataract, if they are to give the best late results.

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2. Jackson, E.: Tr. Am. Ophth. Soc. **9**:145, 1900.

ABSTRACT OF DISCUSSION

Dr. Allen Greenwood, Boston: It is difficult to cover this subject without some reference to the type of operation and the early results. It is apparent that if the eye is healthy prior to an operation for cataract and there are no accidents at the time of operation and no complications during the healing process the late result is almost uniformly good, especially if one limits oneself to consideration of the results present six months or more after the operation. I have a number of patients who were operated on from fifteen to twenty years ago who had uncomplicated cataracts removed without any accident or complications and who still have the vision which they had shortly after the operation. In view of the fact that the majority of operations for cataract are done on patients who have reached and gone beyond their three score years and ten, many of them do not live long enough to have their late results evaluated. The majority of patients who have had uncomplicated operations rarely need to change the final glass given to them.

The ordinary ocular disturbances of elderly people should not be considered as late results of the extraction. Of course, hemorrhages and conditions that accompany diabetes and renal disease, arteriosclerosis and senile choroiditis may occur. Loss of vision produced by such conditions can hardly be listed among the late results of extraction of cataract. Barring such conditions, it has been my experience that after a year has elapsed since the operation, complications such as separated retina, glaucoma and iridocyclitis are rare. When, however, one takes into account patients who do badly at the time of the operation or have disturbing complications immediately after, one may find after the first six months a lessening of vision. Patients with a portion of the iris or bits of capsule adherent to the wound and those who have lost considerable vitreous may at any time have postoperative complications.

After looking over my records I have come to the unexpected conclusion that patients who have had extraction of the lens in the capsule in a healthy eye are in after years somewhat better off than those who have had an extracapsular operation, in spite of the fact that many of these patients have marked iridodonesis. Several times I have operated on a fully mature cataract in one eye by the extracapsular method and shortly after have removed the less mature lens from the other eye by the intracapsular method.

During a period of two and a half years forty-five operations for cataract were performed; the late results of these were good, and the patients had resultant useful vision, which they have retained, except in three cases.

To summarize: Given a healthy eye, a healthy body, no accidents at the time of extraction and no complications during the process of healing, the late results of extraction of cataract should be almost uniformly good, as far as actual results of the operation are concerned.

Given a sick eye, or one previously diseased, a sick body, accidents at the operation or complications following it or during healing, the late results are very uncertain, but even in such cases they may be surprisingly good.

DR. EDWARD C. ELLETT, Memphis, Tenn.: It is a matter of great concern to persons who undergo an operation for cataract as to what their prospects are for retaining the vision which they regain by the operation. I have made a survey of my private patients with cataract whom I was able to examine or have examined after an interval of five years after the operation, and I have reliable data on one hundred and eighty-eight such patients. Only those were considered in whom vision of 6/9 or better was obtained at the time of dismissal. It would not be fair to blame the operation for the failure of the restoration and preservation of vision in an eye already affected with glaucoma, chronic uveitis or high myopia.

Of the patients considered in this survey, 112 were operated on from five to ten years before, 67 from ten to twenty years before and 8 from twenty to thirty years before, and 1 was operated on over thirty years before. The ocular health of these patients was just as good at the end of the period which had elapsed since their eyes were subjected to the operation as it was in a similar number of patients of the same age on whom no operation had been performed.

There seem to be two conditions especially that often develop after a successful operation for cataract, which are capable of impairing the vision, namely, glaucoma and macular degeneration, neither of which seems to have any relation to the operation or the condition for which the operation was performed.

Doubtless the macular disease was already beginning before the operation but was hard to detect when the cataract was present. To those who have had the unfortunate experience of having met with such cases, the following statement of an operator of the widest experience, Colonel Wright, of Madras, India (*Am. J. Ophth.* 20: 376, 1937) is comforting: "Posterior-segment lesions are sometimes a cause of disappointment, and are not necessarily appreciated by preoperative tests."

Another distressing condition sometimes seen is retinal detachment.

None of these conditions, in my opinion, is in any way dependent on the cataract or its removal, and my observation leads me to think that the chances for an eye from which a cataract has been successfully removed retaining useful vision as long as the patient lives is just as good as for the eye of any person of similar age and a similar state of health.

In this series there were eleven patients who secured an immediate good visual result and subsequently suffered loss or impairment of vision. The causes of this loss or impairment were as follows: glaucoma (all varieties), in 2 cases; retinal detachment, in 2; atrophy of the optic nerve, in 4; retinal hemorrhages and macular degeneration, in 2, and uveitis, in 1.

DR. EDWARD JACKSON, Denver: May I call attention to the fact that this paper has brought out points that may be more important than what was presented in the paper? The summaries of the two discussers would invite all the members to look into this matter and consider the results of our operations. This is a field of investigation to be occupied legitimately and with the prospect of getting information.

I wish to speak of the operation that was practiced in all these cases. It was done with a knife, the point of which is like that of a Graefe knife, just wide enough to almost complete the corneal section. The peculiarity of the knife is that it has a curve. First I used a knife that was about 4 or 4.5 mm. broad at the broadest point, and completed the

corneal section by pushing forward, but I came to the conclusion later that it was better to use a narrower knife and if there was a slight bridge of tissue to divide that as the knife was withdrawn, pulling the point up to cut back. When one penetrates the cornea, to make the puncture or the counterpuncture, the knife, having a long angle of cutting edge, strikes the cornea obliquely, and little force is required. If one completes the incision with the Graefe knife, which most operators do by making a sawing motion, changing direction two or three times, one pulls up the sclera, particularly if one makes an incision that goes out into the sclera. I thought this caused too much disturbance to the eye.

Fixation was made in the direction that the knife was pointing when it made the puncture and the counterpuncture. The tendency of the eye to rotate before the pressure was made was entirely balanced, and very easily balanced, with little force applied at that point.

Then the incision was made in the capsule. After I tried this procedure and became familiar with it, it seemed to me it was superior to anything else, and I continued to use it. The knife is brought into the corneal section, after it has been dipped in boiling water to sterilize it, and is drawn back until the point is at about the temporal edge of the pupil. Then it is pushed forward in the capsule of the lens, dividing the capsule from that point parallel to incision in the cornea.

The smooth, prompt healing of that sort of incision justifies it, and justifies mentioning it now, forty-seven years after the operation was described, although comparatively few have adopted this form of knife.

Certainly the point that the general structure and the relations of the eyeball should be as little disturbed as possible in the operation for cataract is an important one to have in mind in every manipulation of the instruments that one makes at the time.

A RARE COMPLICATION FOLLOWING APPENDECTOMY

REPORT OF A CASE IN A FIFTY-FOUR YEAR OLD MAN

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It is probable that small cerebral hemorrhages, causing no symptoms, occur not infrequently during general anesthesia. Thus, patients often state that they have noticed some degree of deafness subsequent to being given a general anesthetic. If these statements are correct it must be supposed that hemorrhage in the inner ear has occurred. Cases of cerebral apoplexy such as the one here reported are rare.

REPORT OF CASE

K. T., a 54 year old carpenter, was brought to the hospital in Drammen on March 21, 1937. The past history contained nothing of significance apart from a chronic ailment of the nose and ear. The patient had undergone an operation for a deviated nasal septum on February 18. At that time his blood pressure had measured 150 systolic and 110 diastolic. During the night prior to his admission to the hospital he had been seized with abdominal pain. The diagnosis of acute appendicitis was confirmed on admission. Operation was performed within a few hours, with the patiént under narcosis induced with scopolamine hydrobromide, morphine and ether. The apparatus of Ombrédanne was used, the customary quantity of ether—100 cc.—being poured into the machine. During the operation the patient collapsed and became extremely cyanotic. After some moments recovery occurred, and the operation was continued, with the patient under the influence of an anesthetic administered by use of the open mask. The quantity of ether used in the aforementioned apparatus was not noted.

When the patient awoke it became apparent that he was totally blind. The attending surgeon also noted paresis of the right arm and leg. The next day I examined the patient. He was a heavy plethoric person, and was restless and somewhat disoriented, so that examination was difficult. The grip of the right hand was weaker then that of the'left. The other reflexes could not be satisfactorily examined. Ocular examination revealed complete ptosis of the left upper lid, the right being normal. Each eye was immobile, deviating a little toward the right but not drawn entirely to the canthus. Each pupil was round and moderately dilated and did not react at all to light. The media and fundi were normal. Total amaurosis was present.

The patient soon became increasingly irrational. He died during the following night. Permission for postmortem examination was not secured. A Wassermann test of the blood or spinal fluid was not made.

In summary, a stout man with a somewhat increased blood pressure showed symptoms of a cerebral hemorrhage with resulting total blindness, oculomotor paralysis and, possibly, paralysis of the left abducens nerve.

COMMENT

The cerebral hemorrhage must have been situated in the primary subcortical visual centers on the left side. As will be remembered, this lies in the region of the pulvinar of the optic thalamus, the lateral geniculate bodies and the anterior corpora quadrigemina, below and a little lateral to the aqueduct of Sylvius. In addition, the nerve fibers involved in the reactions of the pupil, in accommodation and in convergence, were affected.

As the ptosis appeared only on the left, the left oculomotor nucleus alone could be damaged. As the pupil of the right eye was also dilated and failed to react to light, the nerve fibers connecting with the right third nucleus must have been injured. As there was no demonstrable vertical deviation of the eyes, a lesion of either trochlear nucleus can be eliminated. On the other hand, a lesion involving the abducens nerve cannot be eliminated. On this account one cannot dismiss the possibility that the hemorrhage was basal and meningeal in origin. It is more reasonable, however, to assume a pontile localization because of the conjugate deviation of the eyes.

So much may be deduced from the position of the eyes and the pupils. Unfortunately, it cannot be definitely stated how far forward or backward the hemorrhage extended. It is, however, unthinkable that a hemorrhage extending posteriorly could reach so far as to involve both optic radiations, with complete blindness as a result, at any rate, without causing death sooner than it occurred in this case. It is easier to suppose that the bleeding extended straight ahead toward the chiasm, destroying its function.

I have been unable to find in the ophthalmologic literature at my disposal a decription of a similar complication following narcosis induced with ether.[1] Poulsson emphasized the fact that chloroform causes a considerable decrease in the general vascular tonus. Ether, on the other hand, is followed by only a slight change in the general blood pressure, apparently only the centers controlling the blood pressure of the face and the surface of the brain being involved.

It is known that narcosis induced with the use of Ombrédanne's apparatus causes temporary asphyxia. It was probably during this state that rupture of a vessel occurred at some point of diminished resistance in the pons. Too pronounced vasodilatation must have taken place, as was indicated by the severe cyanosis. The resulting hemorrhage affected both the oculomotor and the visual centers.

The case is from the service of Dr. Knud Nicolaysen, who permitted me to make the report.

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1. Search has included Wilbrand and Saenger's "Neurologie des Auges," 1900-1927, Nelson's loose leaf system, many numbers of the *Klinische Monatsblätter*, the *Zentralblatt der Ophthalmologie* and the *Zentralblatt für Chirurgie*, and the tenth edition of Poulsson's "Lehrbuch der Pharmakologie."

BACTERIAL FACTORS IN CHRONIC CATARRHAL CONJUNCTIVITIS

I. RÔLE OF TOXIN-FORMING STAPHYLOCOCCI

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The etiology of chronic catarrhal conjunctivitis is not clearly defined. With no single causative factor in evidence, a variety of agents and conditions have been regarded as inciting or contributing causes. Among the more important of these are the following: (1) bacteria, principally Haemophilus lacunatus (the diplobacillus of Morax and Axenfeld); (2) viruses, such as those causing molluscum contagiosum and the common wart; (3) acne rosacea; (4) allergy; (5) streptotrichal concretions of the canaliculi; (6) trichiasis; (7) minute foreign bodies; (8) irritants, such as smoke; (9) refractive errors; (10) excessive secretion of the meibomian glands; (11) vitamin deficiency; (12) deficient lacrimal secretion, and (13) chronic nasal sinusitis.

It is proposed in this series of studies to consider the bacterial factors in chronic catarrhal conjunctivitis and to endeavor to determine the relationship of each bacterium to the disease. The term chronic catarrhal conjunctivitis will be used to include all forms of chronic conjunctivitis which cannot be placed in any other category.

RELATION OF BACTERIA TO CHRONIC CATARRHAL CONJUNCTIVITIS

The only universally recognized bacterial cause of chronic catarrhal conjunctivitis at present is H. lacunatus (the diplobacillus of Morax and Axenfeld). Its etiologic rôle has been established conclusively by the inoculation experiments on man of Morax,[1] Axenfeld,[2] Hoffmann,[3] Gifford[4] and Erdmann.[5] That the conjunctivitis is due to the liberation

1. Morax, V.: Ann. Inst. Pasteur 10:337, 1896; Ann. d'ocul. 117:5 (Jan.) 1897.

2. Axenfeld, T.: Bacteriology of the Eye, translated by Angus MacNab, London, Baillière, Tindall & Cox, 1908, p. 171.

3. Hoffmann: Arch. f. Ophth. 48:639, 1899; cited by Axenfeld.[2]

4. Gifford, H.: Ann. Ophth. 7:218 (April) 1898; Ophth. Rec. 14:511, 1905; cited by Axenfeld.[2]

5. Erdmann: Klin. Monatsbl. f. Augenh. 63:501, 1905; cited by Axenfeld.[2]

of soluble toxic products of the bacterium is indicated from the experiments of Morax and Elmassian.[6] These investigators induced conjunctivitis by instilling toxins, produced in culture by the diplobacillus, into the conjunctival sacs of animals and human volunteers. That the toxin must also be liberated in the sac in the natural disease may be deduced from the work of Lindner,[7] Howard[8] and others, who have shown that the diplobacilli do not invade the epithelium but proliferate only in the secretion and on desquamated epithelium. The researches of Erdmann[5] and others have shown, further, that diplobacilli may occasionally occur on the healthy conjunctiva. Thus two important principles would seem to have been established: (1) A bacterium multiplying only in the secretion can induce conjunctivitis by the liberation of toxin and (2) a known conjunctivitis-producing bacterium may occur occasionally on the healthy conjunctiva.

The occurrence of many other varieties of bacteria has been noted in chronic catarrhal conjunctivitis. Some of these, such as Corynebacterium xerosis and other diphtheroids, have been generally considered to be without pathogenic significance. To such well known pathogens as Streptococcus haemolyticus and Haemophilus influenzae, on the other hand, an etiologic rôle has frequently been ascribed, although there have been no substantiating results in inoculation experiments on man.

THE BACTERIAL FLORA IN CHRONIC CATARRHAL CONJUNCTIVITIS

A bacteriologic analysis[9] was made in 399 cases of chronic catarrhal conjunctivitis observed in the ophthalmic clinic of the University Hospitals, Iowa City, and of 244 cases observed at the Vanderbilt Clinic, New York. Only those bacteria considered to be of possible etiologic importance are recorded.

In the New York series the relatively small number of cases in which nonpathogenic bacilli were observed was probably due to the repeated cultures which were made in many instances and to the inclusion of white staphylococci, when hemolytic, in the list of etiologic possibilities.

Noteworthy was the failure to find any cases of diplobacillary conjunctivitis in the New York series. Three strains of diplobacilli morphologically like the bacillus of Morax and Axenfeld proved on culture to be distinct from it but were not identified.

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6. Morax, V., and Elmassian: Ann. d'ocul. **72**:6, 1899.
7. Lindner, K.: Ztschr. f. Augenh. **42**:11, 1919; Arch. f. Ophth. **55**:726, 1921.
8. Howard, H. J.: Tr. Am. Ophth. Soc. **22**:186, 1924.
9. Cultures made on blood agar by inoculation of material obtained from the lower fornix with a platinum loop were incubated at 37 C. and examined after forty-eight hours. In the New York series sterile swabs moistened in broth containing 1 per cent dextrose were used in taking material.

The staphylococcus is seen to be by far the most common bacterium of possible etiologic consequence observed in chronic conjunctivitis in these series, and its significance is the subject of this first report. ·

TABLE 1.—*Bacteria Noted in Cases of Chronic Catarrhal Conjunctivitis*

Iowa Series	
Bacteria	No. of Cases
Staphylococcus aureus	175
Diplococcus pneumoniae	22
Haemophilus lacunatus (Morax, Axenfeld)	9
Neisseria catarrhalis	4
Streptococcus haemolyticus	4
Streptococcus viridans	2
Haemophilus influenzae	2
Staphylococcus aureus and Haemophilus lacunatus	3
Staphylococcus aureus and Haemophilus influenzae	1
Staphylococcus aureus and Diplococcus pneumoniae	5
Staphylococcus aureus and Streptococcus viridans	1
Staphylococcus aureus and Streptococcus haemolyticus	2
Staphylococcus aureus, Diplococcus pneumoniae and Neisseria catarrhalis	1
Haemophilus lacunatus and Diplococcus pneumoniae	3
Haemophilus influenzae and Aerobacter aerogenes	1
Unidentified gram-negative bacilli	2
Nonpathogenic bacteria (including diphtheroids, Staphylococcus albus and sarcinae)	162
Total	399

New York Series	
Staphylococcus aureus	80
Staphylococcus albus (hemolytic)	78
Diplococcus pneumoniae	6
Haemophilus influenzae	5
Streptothrix Foersteri	2
Streptococcus haemolyticus	1
Proteus ammoniae	3
Staphylococcus aureus and Diplococcus pneumoniae	1
Staphylococcus aureus and Staphylococcus albus (hemolytic)	9
Staphylococcus aureus and Streptococcus viridans	2
Staphylococcus aureus and Streptococcus haemolyticus	1
Staphylococcus albus (hemolytic) and Diplococcus pneumoniae	1
Staphylococcus albus (hemolytic) and Streptococcus nonhaemolyticus	2
Haemophilus influenzae and Aerobacter aerogenes	1
Pseudomonas aeruginosa, Escherichia coli and Klebsiella pneumoniae	1
Unidentified gram-negative bacilli	3
Nonpathogenic bacteria (including diphtheroids, nonhemolytic Staphylococcus albus and sarcinae)	48
Total	244

ARE STAPHYLOCOCCI PATHOGENIC FOR THE CONJUNCTIVA?

History.—Negative results of inoculation experiments with staphylococci on the human conjunctiva have been reported by Leber,[10] Sattler,[11]

10. Leber: Internat. Cong. Ophth., Heidelberg, 1880; cited by Axenfeld,[2] p. 233.

11. Sattler: Internat. Cong. Ophth., Heidelberg, 1880; cited by Axenfeld,[2] p. 233.

Bach [12] and Hirota.[13] A single inoculation with a positive result has been performed by McKee.[14] Morax and Elmassian [6] have demonstrated conjunctivitis-producing toxin in broth cultures of Staphylococcus aureus isolated from the conjunctiva. When instilled into the eyes of human volunteers and of laboratory animals this toxin produced a definite conjunctival inflammation after an incubation period of a few hours. These results would seem to be of primary importance, but in the absence of confirmatory reports some observers, including Lindner [7] and Howard,[8] stated the opinion that staphylococci appear on the conjunctiva only as harmless saprophytes. Others, such as Burky,[15] have expressed the belief that they are an important cause of conjunctivitis.

Inoculation Experiments.—In view of the difficulty which is encountered in interpreting the results of inoculation experiments on the conjunctiva, it would seem that in the case of staphylococci the following conditions for a significant experiment should be satisfied: (1) A virulent, freshly isolated strain must be employed and (2) the staphylococci must multiply on the conjunctiva. The second condition is of prime importance, since staphylococci are known to multiply in conjunctivitis, and any inoculation experiment in which they are rapidly eliminated from the conjunctiva should be considered of little or no significance. Inoculations in man are far more valuable than inoculations in animals, on account of the resistance of laboratory animals to bacterial conjunctivitis.

In the absence of human volunteers a series of inoculations with freshly isolated toxigenic staphylococci from chronic conjunctivitis were made on rabbits, guinea-pigs and monkeys, in groups of six. These animals proved to be wholly resistant to simple deposition of cultures on the conjunctiva.

In a second series, instillations in monkeys and rabbits, followed by vigorous massage with cotton applicators, produced moderate conjunctivitis that persisted for several days. Control eyes, treated by massage alone, showed only temporary irritation.

A third series of rabbits and monkeys were subjected to chemical irritation by instillations of a 2 per cent solution of silver nitrate. Two hours later one eye of each animal was heavily inoculated with staphylococci. The resultant conjunctivitis was much more marked in the inoculated eyes and persisted for several days after the irritation of the uninoculated eyes had healed. In no instance, however, did chronic conjunctivitis develop.

In a fourth series of rabbits fresh cultures were injected intracorneally. The corneal abscesses or ulcers which resulted were always accompanied by a severe purulent conjunctival reaction.

12. Bach, L.: Arch. f. Ophth. **40**:3, 1894; cited by Axenfeld,[2] p. 233.

13. Hirota, cited by Axenfeld,[2] p. 233.

14. McKee, S. H.: Bacteriology of the Eye, in Wood, C.: American Encyclopedia of Ophthalmology, Chicago, Cleveland Press, 1913, vol. 2, p. 824.

15. Burky, E. L.: Am. J. Ophth. **19**:841, 1936.

The results in these four series of experiments indicate that under certain circumstances staphylococci may be pathogenic for the conjunctiva of laboratory animals, but no chronic conjunctivitis comparable to that occurring in man could be produced, and it was impossible to establish growth of staphylococci in the conjunctiva; the bacteria were always rapidly eliminated.

Toxin Conjunctivitis.—One hundred and fifty strains of staphylococci from material from eyes with chronic conjunctivitis were tested for conjunctivitis-producing exotoxin. The toxins were prepared according to the method of Leonard and Holm,[16] according to which growth is maintained in semisolid agar in an atmosphere of 80 per cent carbon dioxide and 20 per cent oxygen. They were adjusted to a p_H of from 6.8 to 7 and rendered bacteria-free by filtration through Mandler filters.

The conjunctivitis-producing power was determined by instillation of filtrates into the eyes of adult white rabbits at five minute intervals for one hour. Of the 150 filtrates tested, 57 exhibited definite conjunctivitis-producing power. The test conjunctivitis was characterized by (1) an incubation period varying from one to two hours and (2) a purulent inflammation varying in degree from a mild but definite conjunctivitis to an acute reaction with swelling of the lids and closure of the eye. The conjunctivitis subsided in two or three days, leaving no demonstrable immunity.

Two tests on the human conjunctiva showed it to be apparently more susceptible to the toxin than the conjunctiva of the rabbit, a single drop of active toxin producing acute or subacute conjunctivitis with bulbar hemorrhages.

The following evidence indicated that the test conjunctivitis was a true toxic reaction: (1) The uninoculated medium produced no irritation; (2) the toxin, when adjusted to neutrality, produced no immediate signs of inflammation; (3) there was a definite period of incubation; (4) toxin detoxified by formaldehyde, boiling or prolonged alkalinization did not produce conjunctivitis, and (5) immunization by intracutaneous injections of toxin weekly for ten weeks prevented toxin conjunctivitis in two tests on rabbits. (Immunization was not effected by repeated local instillations of toxin.)

Evidence in Favor of the Existence of Staphylococcic Conjunctivitis.—The evidence from this study which indicates that staphylococci are a factor in the production of conjunctivitis may be summarized as follows: 1. Virulent staphylococci have been observed in material from eyes with conjunctivitis in which no other cause could be demonstrated. Representative strains have proved to be pathogenic for rabbits on

16. Leonard, G. F., and Holm, A.: J. Immunol. **29**:209, 1935.

intracorneal and intravenous injection, and toxins produced by them have shown the hemolytic, dermonecrotic and lethal properties characteristic of toxins of pathogenic staphylococci from other sources. 2. A conjunctivitis-producing factor has been demonstrated in toxins from these bacteria. Fifty-seven of 150 strains of Staphylococcus aureus and Staphylococcus albus from inflamed conjunctivae produced toxin causing conjunctivitis, and there was a positive correlation between the toxicity of strains and their source. Thus, the majority of staphylococci which occurred in large numbers in the initial cases of conjunctivitis and were presumed to be its cause were toxin-producers, whereas those present in insignificant numbers were generally nontoxic. 3. Positive cultures were obtained throughout the duration of the disease in 17 cases of conjunctivitis in which staphylococci were the only significant bacteria present. 4. Disappearance of the staphylococci after healing of the disease occurred in all but 3 of the 17 cases. In these 3 the bacteria persisted but were greatly reduced in number. 5. Staphylococcus toxoid [17] was effective in healing the condition in 13 of the 17 cases after failure of local treatment.

Delimitation of Staphylococcic Conjunctivitis.—The frequency of staphylococci on the inflamed conjunctiva and the difficulty of distinguishing pathogenic from nonpathogenic strains have made it difficult to delimit staphylococcic conjunctivitis. The only way of making an absolute diagnosis found in this study was to observe a case until healing occurred. A presumptive diagnosis could be made when large numbers of staphylococci of the aureus variety were present in eyes with conjunctivitis which had no other demonstrable cause, and the probability of its correctness was increased by the presence of the characteristic lesions of blepharitis or recurrent marginal ulceration of the cornea. The greatest difficulty was experienced in the evaluation of the rôle of the white staphylococci, for this study has shown that certain albus strains are capable of forming conjunctivitis-producing toxin.

Clinical Characteristics of Staphylococcic Conjunctivitis.—In the New York series there were 17 cases observed until healing occurred in which toxin-producing staphylococci were considered to be the sole cause and 37 additional cases in which staphylococci were considered to be the cause but which could not be observed to the stage of healing. Among the forms of conjunctivitis in these 54 cases were the following four clinical types: (1) conjunctivitis with an acute or a subacute onset and a definite tendency to become chronic; (2) chronic conjunctivitis with recurrent catarrhal ulcers; (3) chronic blepharoconjunctivitis, and (4) chronic catarrhal conjunctivitis without other involvement.

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17. The Lederle Laboratories, New York, supplied the toxoid used in this study.

The most common form appeared to be blepharoconjunctivitis. All degrees of it were seen, from simple hyperemia of the margin of the lid to ulcerative blepharitis, which was associated in one case with sycosis vulgaris.

Staphylococcic conjunctivitis was found to have the following cardinal characteristics: (1) a protracted course, (2) resistance to local therapy and (3) frequency of concomitant blepharitis and ulceration of the margin of the cornea.

Staphylococcic conjunctivitis of the adult was found to differ markedly from the condition in the new-born (the latter has been reported separately [18]), in whom the disease was always acute or subacute, healed spontaneously or with simple antiseptic therapy and was not accompanied by involvement of the margin of the lid or by corneal involvement.

Correlation of Toxin Production of Staphylococci with Source from Which Isolated.—Staphylococci from the following sources were analyzed for the production of toxin: I. Eyes with conjunctivitis observed to healing in which staphylococci could be certainly identified as the causal factor. II. Eyes with conjunctivitis of which staphylococci were probably the cause but which could not be observed to healing. III. Eyes with conjunctivitis of which staphylococci were certainly not the sole cause but were probably a contributory cause. Included among these cases were instances of specific disease, such as trachoma, and cases of unknown etiology in which the conjunctivitis continued after disappearance of the staphylococci. IV. Eyes with conjunctivitis in which staphylococci were certainly not the cause and in which they were present in too small numbers or too irregularly to be significant.

Nonhemolytic white staphylococci were omitted from consideration in this analysis but will be discussed in a later report.

From source I, Staph. aureus was isolated in 15 cases and Staph. albus in 2. The aureus strains all produced soluble hemolysin in titers of 1:10 or greater, and all but 4 formed toxins having definite conjunctivitis-producing power. The two albus strains showed neither soluble hemolysin nor conjunctivitis-producing toxin.

From source I, Staph. aureus was isolated in 15 cases and Staph. albus in 22. All but 1 of the aureus strains showed soluble hemolysin, and 27 of the 37 showed conjunctivitis-producing toxin. Only 9 of the albus strains showed hemolysin and only 4 conjunctivitis-producing toxin.

From source III, Staph. aureus was isolated in 22 cases and Staph. albus in 24. Of the aureus strains, 16 showed soluble hemolysin and 12

18. Thygeson, P.: Tr. Am. Ophth. Soc. **34**:340, 1936.

conjunctivitis-producing toxin. Only 1 albus strain produced soluble hemolysin and only 3 conjunctivitis-producing toxin.

From source IV, Staph. aureus was not isolated, and Staph. albus was isolated in 26 cases. In none of these did Staph. albus show soluble hemolysin or conjunctivitis-producing toxin.

Thus there appeared to be a definite correlation between the source and the production of toxin, sources I and II providing a high percentage of toxin-producing strains and source IV none.

PROPERTIES OF STAPHYLOCOCCI FROM EYES WITH CONJUNCTIVITIS

The determination of the pathogenicity of a given strain of staphylococci is a time-consuming laboratory procedure requiring inoculation of animals. Many investigators have attempted to find more simple means of estimating this factor, and an apparent correlation with toxicity has been noted among the following properties of the bacterium: (1) hemolytic activity on blood agar plates, (2) production of pigment, (3) liquefaction of gelatin, (4) ability to coagulate blood plasma, (5) ability to ferment mannitol and (6) ability to produce violet growths on crystal violet agar.[19] An attempt has been made in this study to determine whether or not a correlation exists between any of these easily determined properties and the probable pathogenicity of the strain for the conjunctiva as determined by the source from which the strain was isolated and its ability to form conjunctivitis-producing and hemolytic toxins.

Staphylococci Showing Conjunctivitis-Producing Toxin.— Table 2 summarizes some of the biologic properties of staphylococci, the toxins of which were found to be conjunctivitis-producing when tested on rabbits' eyes. Their biologic activity was marked. Of the 57 strains (50 aureus and 7 albus), all but 1 produced hemolysis on rabbit blood agar plates, all but 5 produced soluble hemolysin in a titer of 1:10 or more, all but 4 coagulated human blood plasma, all but 5 fermented mannitol, and all but 9 produced a purple growth on crystal violet agar. Of the 40 strains tested for liquefaction of gelatin, 22 gave positive results.

Staphylococci Showing Soluble Hemolysin but Questionable or No Conjunctivitis-Producing Toxin.—As indicated in table 3, these strains were also active biologically. All 26 strains (22 aureus and 4 albus) produced hemolysin on plates and soluble hemolysin; all coagulated human blood plasma; all but 2 fermented mannitol, and all but 4 produced purple growths on crystal violet agar. All but 10 liquefied gelatin.

19. Chapman, G. H., and Berens, C.: J. Bact. **29**:437, 1935.

Strain	Pigment	Hemolysin on Plates	Soluble Hemolysin, Titer	Coagulase	Liquefaction of Gelatin	Fermentation of Mannitol	Purple Growth on Crystal Violet Agar	Conjunctival Reaction
5	Aureus	++	1:1,000	+++	—	++	+	+
19	Aureus	++++	1:1,000	++++	++++	++	+	+
21	Aureus	++++	1:1,000	+++	+++	+++	+	+
28	Aureus	++++	1:100	++	—	+++	+	+
33A	Aureus	+++	1:10	++++	—	+++	+	++
34	Aureus	++++	1:10	++++	—	+++	+	++++
40A	Aureus	+++	1:1,000	++	—	—	+	++
43	Aureus	++++	1:10	++++	—	++	+	++
46	Aureus	+	1:1,000	+++	—	+++	+	+++
51	Aureus	++++	1:10	+++	—	+	+	+++
54A	Aureus	+	Negative (1:10)	+++	++	+++	+	++++
56	Aureus	++	1:1,000	+++	++	+++	+	++++
63	Aureus	++++	1:1,000	++++	++++	++	+	++
72	Aureus	+++	1:10	++++	—	++	+	+
76	Aureus	++++	1:100	++++	++++	+	+	++++
84A	Aureus	++++	1:1,000	+++	++++	++++	+	+++
105	Aureus	++	1:100	++++	++++	++++	+	+
109	Aureus	++++	1:100	++++	++++	++++	+	+
113	Aureus	++++	1:100	++++	+++	++++	+	+
18	Aureus	+++	1:1,000	++++	+	++++	+	++++
68B	Aureus	++	1:100	++++	+	+++	+	+
126	Aureus	+++	1:1,000	+++	—	++++	+	+
87A	Aureus	....	1:100	++++	+	++++	—	+
87B	Aureus	+++	1:100	+++	—	—	—	+
136	Aureus	++++	1:1,000	++++	++++	++	+	++
146	Aureus	....	1:1,000	+++	—	+—	+	+
159	Aureus	++	1:100	++++	+++	+++	+	++++
11	Aureus	++++	1:1,000	++++	++++	+	+	++++
158A	Aureus	++++	1:100	++++	+	++	+	+++
161	Aureus	++++	1:10	++++	++++	+++	+	+
168B	Aureus	—	1:100	++++	—	++	+	+
197	Aureus	+++	1:100	+++	+++	++++	+	+++
237	Aureus	+	1:10	++	—	++++	+	+++
238	Aureus	++++	1:100	+++	++++	+	+	+
239	Aureus	+++	1:100	+++	++++	+	+	+
271	Aureus	++	1:100	+++	++++	++	+	+
283	Aureus (delayed)	++++	1:1,000	+++	....	+++	+	++
284	Aureus	++	1:1,000	++++	....	++++	+	++++
288	Aureus	+	1:100	+++	....	++++	+	+
290	Aureus	+	1:1,000	+++	....	++++	+	+
291	Aureus	+++	1:1,000	++++	....	++++	+	+
292	Aureus	+++	1:1,000	+++	....	++++	+	++++
294	Aureus	+	1:10	—	....	++++	+	+
295	Aureus	+	1:10	+++	....	++++	+	+
296	Aureus	±	1:100	++	....	++++	—	++++
301	Aureus	+	1:100	+++	....	++++	—	+
308	Aureus	++	1:100	++++	....	++	+	+++
311	Aureus	+	1:1,000	++++	....	++++	+	+
335	Aureus	++++	1:1,000	++	....	+++	—	+
341	Aureus	+	1:1,000	+++	....	++++	+	+
32	Albus	++	Negative (1:10)	++++	—	—	—	+
45	Albus	++	Negative (1:10)	—	—	—	—	+
106	Albus	++	1:1,000	++++	—	++	+	+
81B	Albus	+	Negative (1:10)	—	—	—	+	+
289	Albus	++++	1:1,000	++++	....	++++	+	+
322	Albus	+++	Negative (1:10)	—	....	+	—	++
340	Albus	++	1:1,000	+	....	++++	+	++

* The pigment was estimated after forty-eight hours' growth on plain agar. Hemolysin on plates was estimated on rabbit blood agar plates at the time of isolation of the strain after forty-eight hours of incubation. In the coagulase test, one loopful of a twenty-four hour culture on a solid medium was mixed with 0.5 cc. of citrated human plasma and incubated for three hours at 37 C. In the test for liquefaction of gelatin, the results were estimated after three weeks of incubation at 37 C. In the test for fermentation of mannitol, readings were taken after forty-eight hours. In the test with crystal violet agar, the reaction was considered positive if a violet or partly violet growth was present after thirty-six hours of incubation. The conjunctival reaction was determined on adult white rabbits. Toxin was instilled at five minute intervals for one hour, and readings were taken after twenty-four hours. + indicates a mild but definitely purulent conjunctivitis; ++++ indicates a severe purulent conjunctivitis with closure of the eye due to swelling of the lids. To eliminate faulty technic in the preparation of toxins as a factor in this test, a known toxin producer was included as a control with each group of strains studied for the production of toxin.

Staphylococci Showing Neither Soluble Hemolytic Toxin nor Conjunctivitis-Producing Toxin.—Table 4 summarizes the more important properties of the 57 strains (6 aureus and 51 albus) which produced neither soluble hemolysin nor conjunctivitis-producing toxin. They were found to be relatively inactive biologically. All hemolyzed rabbit blood agar to some degree; only 2 strains coagulated human blood plasma; only 9 fermented mannitol, and only 3 produced a purple growth on

TABLE 3.—*Properties of Staphylococci Showing Soluble Hemolysin but Questionable or no Conjunctivitis-Producing Toxin*

Strain	Pigment	Hemolysin on Plates	Soluble Hemolysin, Titer	Coagulase	Liquefaction of Gelatin	Fermentation of Mannitol	Purple Growth on Crystal Violet Agar	Conjunctival Reaction
55	Aureus	++	1.10	++++	±	++++	+	—
70A	Aureus	+	1:10	++++	++++	++	+	±
78A	Aureus	+++	1:100	++++	—	++++	—	—
4	Aureus	++++	1:100	+++	++++	++++	+	—
80	Aureus	++++	1:1,000	+++	+++	+++	+	—
98	Aureus	+++	1:100	++	—	++++	+	±
112	Aureus	++++	1:1,000	++++	—	+	+	±
138	Aureus	++	1:10	++++	—	++++	—	±
117	Aureus	+++	1:10	++++	+	++++	+	—
142	Aureus	++	1:100	++++	—	++	+	—
149	Aureus	++	1:10	++++	+++	++	+	—
151	Aureus	++	1:100	++++	—	+++	+	±
173	Aureus	+++	1:100	++++	—	+++	+	±
175	Aureus	++++	1:1,000	++++	++++	++++	+	—
206	Aureus	++++	1:10	++++	+	++	+	—
232	Aureus	++	1:1,000	++++	—	—	—	—
243	Aureus	+++	1:100	+++	—	++++	+	—
250	Aureus	+++	1:100	+++	—	+	+	±
246	Aureus	++	1:1,000	++++	++++	+++	+	—
253	Aureus	+++	1:1,000	++++	—	++++	+	—
280	Aureus	+++	1:100	++++	++++	++	+	—
143	Aureus	++	1:10	++	....	++++	+	—
50	Albus	+++	1:10	++	—	+	+	±
150	Albus	++++	1:1,000	++++	++++	++++	+	—
231	Albus	+++	1:1,000	+++	—	—	—	—
293	Albus	++	1:1,000	++++	....	++++	+	±

crystal violet agar. Of the 52 strains tested for liquefaction of gelatin, only 6 gave positive results.

Biologic Tests as Indicators of Toxicity.—There was no absolute correlation between the production of toxin and any of the properties tested (table 5). The closest correlation was with the coagulase test: There was a correlation of 92.9 per cent between it and the production of conjunctivitis-producing toxin and of 100 per cent between it and the production of soluble hemolysin. Only 3.5 per cent of the strains which appeared to be nontoxic gave positive reactions to the coagulase test. It would thus appear that strains that gave positive reactions to the

TABLE 4.—*Properties of Staphylococci Showing Neither Soluble Hemolysin nor Conjunctivitis-Producing Toxin*

Strain	Pigment	Hemolysin on Plates	Soluble Hemolysin, Titer of 1:10	Coagulase	Liquefaction of Gelatin	Fermentation of Mannitol	Purple Growth on Crystal Violet Agar	Conjunctival Reaction
25A	Aureus	++	—	—	++++	—	÷	—
69	Aureus	++++	—	+++	—	+++	÷	—
95	Aureus (delayed)	±	—	—	÷	—	—	—
66	Aureus	++÷	—	—	—	—	—	—
68A	Aureus	÷	—	—	—	—	—	—
73B	Aureus (delayed)	+++	—	—	÷÷÷	—	—	—
1	Albus	++÷	—	—	—	—	—	—
25B	Albus	÷+	—	—	—	—	—	—
27	Albus	÷	—	—	+++	—	—	—
37	Albus	÷	—	—	—	—	—	—
40B	Albus	+	—	—	—	—	—	—
48	Albus	+++	—	—	—	—	—	—
52	Albus	++	—	—	—	—	—	—
57	Albus	÷	—	—	—	—	—	—
58	Albus	+	—	—	—	—	—	—
59	Albus	++	—	—	—	—	—	—
62	Albus	±	—	—	—	—	·	—
64	Albus	++	—	+++	—	—	—	—
65	Albus	++	—	—	—	—	—	—
53	Albus	++	—	—	—	—	—	—
73A	Albus	+++	—	—	—	—	—	—
75	Albus	+	—	—	—	—	—	—
77	Albus	+++	—	—	—	—	—	—
78B	Albus	++++	—	—	—	·—	—	—
85A	Albus	+++	—	·	—	—	—	—
85B	Albus	+++	—	—	—	—	—	—
91	Albus	++	—	—	—	—	—	—
83	Albus	++++	—	—	—	—	—	—
92	Albus	++	—	—	—	—	—	—
94	Albus	++++	—	—	— ·	—	—	—
107	Albus	+++	—	—	—	—	—	—
115	Albus	++	—	—	—	—	—	—
81A	Albus	÷	—	—	—	—	—	—
81B	Albus	÷	—	—	—	—	—	—
116	Albus	÷	—	—	÷	—	—	—
121	Albus	+	—	—	—	—	÷	—
135	Albus	++++	—	—	—	—	—	—
152	Albus	+++	—	—	—	—	—	—
153	Albus	+++	—	—	—	—	—	—
120	Albus	++	—	—	—	—	—	—
138B	Albus	+++	—	—	—	—	—	—
163	Albus	++	—	—	—	—	—	—
178	Albus	+++	—	—	—	—	—	—
221	Albus	....	—	—	—	—	—	—
224	Albus	+++	—	—	—	÷++	—	—
234	Albus	++	—	—	—	—	—	—
241	Albus	++++	—	—	—	÷	—	—
244	Albus	++++	—	—	—	÷	—	—
247	Albus	+++	—	—	—	—	—	—
266	Albus	++	—	—	÷÷÷	—	·	—
268	Albus	+++	—	—	—	—	—	—
267	Albus	++	—	—	—	—	—	—
304	Albus	++++	—	—	—	÷÷	—	—
318	Albus	++++	—	—	..	÷	—	—
320	Albus	÷	—	—	..	÷	—	—
326	Albus	++	—	—.	..	÷÷	—	—
327	Albus	÷+	—	—	..	—	—	—

coagulase test would in most instances be toxic, particularly if pigment-forming. The more simple tests, those with crystal violet agar and the fermentation of mannitol, would appear to have corroborating value as indicators, even though less reliable than the coagulase test. Liquefaction of gelatin, on the other hand, was of no apparent value as an indicator of toxicity.

Is Conjunctivitis-Producing Toxin Identical with Hemolytic Toxin?—Staphylococcus toxin is known to be hemolytic, particularly for rabbit cells, to have a destructive action on leukocytes, a necrotic action when injected intradermally in rabbits, a lethal action when injected

TABLE 5.—*Correlation of Biologic Properties with Production of Toxin*

	No. of Strains	Percentage
Conjunctivitis-Producing Toxin		
Pigment	50/57	87.7
Coagulase	53/57	92.9
Fermentation of mannitol	52/57	91.2
Purple growth on crystal violet agar	48/57	84.2
Liquefaction of gelatin	22/40	55.0
Soluble Hemolysin but Questionable or No Conjunctivitis-Producing Toxin		
Pigment	22/26	86.1
Coagulase	26/26	100.0
Fermentation of mannitol	24/26	92.3
Purple growth on crystal violet agar	22/26	84.6
Liquefaction of gelatin	10/26	38.4
No Toxin		
Pigment	6/57	10.5
Coagulase	2/57	3.5
Fermentation of mannitol	9/57	15.7
Purple growth on crystal violet agar	3/57	5.2
Liquefaction of gelatin	6/52	11.5

intravenously, and a coagulating action on blood plasma. It has been a matter of some dispute as to whether these actions are the result of a single toxin or of a number of toxins.[20] Many workers, including Burnet,[21] Grosz[22] and Gengou,[23] have based their opinion that only one toxin is formed on the following facts: (1) There has been a fairly constant relationship between the hemolytic, the necrotic, and the lethal powers in different batches of toxin; (2) all manifestations of toxicity are abolished by exposure to a temperature of 56 C. for half

20. Topley, W. W. C., and Wilson, G. S.: Principles of Bacteriology and Immunity, ed. 2, Baltimore, William Wood & Company, 1936.

21. Burnet, F. M.: J. Path. & Bact. **32**:717, 1929.

22. Grosz, H.: Ztschr. f. Immunitätsforsch. u. exper. Therap. **73**:14, 1931.

23. Gengou, O.: Ann. Inst. Pasteur **48**:135, 1932.

an hour, and (3) an antiserum prepared against the toxin neutralizes against all manifestations.

Recent findings, however, point to a multiplicity of toxins. The results of the investigations of Bigger[24] and of Glenny and Stevens[25] suggest that there are at least two hemolysins, and Dolman[26] has failed to find any parallelism between the hemolytic titers of a series of different filtrates against suspensions of red cells of different laboratory animals and concludes that the hemolysins are multiple. Panton and Valentine,[27] furthermore, have found a close relationship in different filtrates between the hemolysin and the necrotoxin but no relation between these and the leukocidin.

Topley and Wilson[20] concluded that the toxin is probably complex but that the hemolysin, the necrotoxin and the lethal toxin are all closely associated. Certain strains of staphylococci considered responsible for outbreaks of food poisoning have been found to give rise to a so-called enterotoxin which has an irritating effect on the intestinal mucous membrane of man and of the monkey. It is heat-resistant, and there seems to be little doubt that it is distinct from the other toxins.

In the present study there seemed to be evidence of a certain relationship between the hemolytic and the conjunctivitis-producing toxins, since only 5 of the 57 strains showing a conjunctivitis-producing toxin failed to give a soluble hemolysin in a titer of 1 : 10 or more. There is some evidence, however, which indicates that the two toxins may be distinct: 1. Of the 57 strains producing conjunctivitis, 5 (1 aureus and 4 albus) failed to produce demonstrable hemolysin. As recorded in table 2, one of these strains that did not produce hemolysin, no. 54 A, produced very severe conjunctivitis. 2. Twenty-six strains (table 3) gave a soluble hemolysin but no definite conjunctivitis-producing toxin. It is true that 9 of the 26 produced conjunctival reactions classed as questionable, but 4 strains (nos. 173, 175, 206 and 231), each tested twice on different animals with toxin from different batches, failed to produce conjunctivitis. 3. As can be seen in table 2, there was no strict parallelism between the potency of the conjunctivitis-producing toxin and that of the soluble hemolysin. For example, of the 10 strains giving a + + + + conjunctival reaction, only 5 gave a soluble hemolysin in a titer of 1 : 10, while 3 gave a soluble hemolysin in a titer of 1 : 100; 1 gave a soluble hemolysin in a titer of 1 : 10, and 1 gave negative results.

24. Bigger, J. W.: J. Path. & Bact. **36**:87, 1933.

25. Glenny, A. T., and Stevens, M. F.: J. Path. & Bact. **40**:201, 1935.

26. Dolman, C. E.: Tr. Roy. Soc. Canada (Sect V.) **26**:309, 1932.

27. Panton, P. N., and Valentine, F. C. O.: Lancet **1**:506, 1932.

Failure of Repeated Attacks of Toxin Conjunctivitis to Immunize.— No evidence was obtained to indicate that local instillations of staphylococcus toxin into the conjunctival sacs of rabbits had any immunizing effect. Thus the right eye of rabbit 136 gave a $++++$ reaction on four successive courses of toxin over a period of one month, and the right eye of rabbit 139 still gave a $+++$ reaction after conjunctivitis had been produced in it six times during a period of seven weeks. A single drop of toxin instilled daily over a period of six weeks into the left eye of rabbit 135 failed to prevent a $++++$ reaction when the usual twelve instillations given at five minute intervals were made.

Relationship of Hemolysin on Plates to Conjunctivitis-Producing Toxin.—As may be seen from examining tables 2, 3 and 4, there was no correlation between the ability of strains of staphylococci to produce hemolysis on rabbit blood agar plates and their ability to form conjunctivitis-producing toxin. Whereas all but 1 of the strains in this study produced hemolysis on blood agar plates, only 57 produced a definite conjunctivitis-producing toxin. Strain 296, for example, which produced only a very faint ring of hemolysis on the plate, caused severe conjunctivitis when its toxin was tested on the rabbit; and table 4 shows that there were 9 strains which showed maximal hemolysis on plates but for which neither soluble hemolysin nor conjunctivitis-producing toxin could be demonstrated in the filtrate.

Staphylococcus Albus as a Cause of Conjunctivitis.—While the majority of strains capable of forming conjunctivitis-producing toxin were of the aureus variety, there were 7 albus strains which produced potent toxins. It is apparent from both the clinical and the laboratory studies that certain strains of Staph. albus should be considered a cause of conjunctivitis. This question will be covered in detail in the second report of this series.

COMMENT

Staphylococcic conjunctivitis resembles diplobacillary conjunctivitis in the following important respects: (1) a protracted course, (2) failure of the bacteria to grow on or in the epithelium, (3) frequency of concomitant blepharitis, and (4) occurrence of occasional acute or subacute cases. The mechanism of production of the conjunctivitis, i. e., by the liberation of toxin in the conjunctival sac by bacteria proliferating in the secretion or on desquamated epithelial cells, is the same for both. Staphylococcic conjunctivitis, however, would seem to be much more difficult to define, owing to the frequent occurrence of saprophytic varieties of staphylococci on both normal and inflamed conjunctivae.

In addition to being the sole cause of chronic catarrhal conjunctivitis in certain cases, pathogenic staphylococci are frequently found secon-

darily complicating specific types of conjunctivitis, notably trachoma and phlyctenular conjunctivitis.

The rôle of albus strains, particularly those that produce hemolysis on rabbit blood agar, is not clear. There is no question but that some of them are active conjunctivitis-producers, but the significance of those from which no conjunctivitis-producing toxin is obtained is obscure. The possibility must be considered that some of these may produce toxin in a quantity insufficient to be measured but sufficient to produce low grade irritation of the conjunctiva.

No evidence was obtained in this study to suggest that allergy to staphylococcus toxin played any part in the reaction of the conjunctiva.

SUMMARY AND CONCLUSIONS

Evidence is advanced to indicate that toxin-producing strains of Staph. aureus and Staph. albus are pathogenic for the conjunctiva.

Staphylococci appear to be a frequent cause of chronic catarrhal conjunctivitis.

The conjunctivitis appears to be due to the liberation of a conjunctivitis-producing toxin, the staphylococci multiplying in the conjunctival secretion or on dead cells but not invading the conjunctival epithelium.

Some of the characteristics of this conjunctivitis-producing toxin are recorded. It is usually produced simultaneously with the hemolytic toxin (α hemolysin) but may be distinct from it.

Of the simple tests which have been proposed for estimating the toxicity of staphylococci, the coagulase test was found to give the highest correlation.

FORMATION OF DRUSEN OF THE LAMINA VITREA

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The ophthalmoscopic appearance of drusen, or "colloid" excrescences on the lamina vitrea, is well known. The changes are seen in elderly persons as numerous bright, sharply circumscribed points with slightly pigmented margins and are usually in the macular zone or around the papilla. In some cases the discrete points become confluent and rather large. The majority of patients with this condition have no visual disturbances, but when the dots are very numerous and fused, the condition can result in diminution of the visual acuity by pressure on the rods and cones that causes their atrophy. Such senile changes early attracted the attention of the pathologists, and H. Müller [1] in 1855 carefully studied them. He found that they were almost constant in persons over 60 years of age, very frequent in persons over 45 and not rare in younger persons. Histologically, they were observed to be most frequent in the neighborhood of the ora serrata and around the optic disk. Since Müller's initial study these colloid bodies have been the subject of considerable investigation, both as a senile manifestation and as a concurrence in pathologic conditions, and many theories have been offered for their origin. The present study is an attempt to review the entire subject and bring it to date, and to offer some observations which would seem to confirm the views of some of the previous investigators.

For an understanding of this condition it is first of all necessary to appreciate Bruch's membrane. This, together with Descemet's membrane and the capsule of the lens, forms a system of glassy membranes peculiar to the eye and resembling no other structures in the body. The origin of these three membranes is varied, however; Descemet's is undoubtedly derived from the mesoderm, while the capsule of the lens apparently is derived from the epithelium of the lens. Bruch's membrane, however, is more complex, and apparently both the mesoderm and the neural ectoderm contribute to its formation. There are not enough

1. Müller, H.: Anatomische Beiträge zur Ophthalmologie, Arch. f. Ophth. (pt. 2) **2**:1, 1855.

embryologic observations to clarify this matter, for at the 5 mm. stage the choriocapillaris first appears, and this rapidly becomes thicker. At the 14 mm. stage it becomes separated from the outer wall of the vesicle by a clear membrane. No more than this is known of its origin in man. Wolfrum[2] has shown in investigations on animals that the basal membrane develops early, almost concurrently with formation of the secondary optic vesicle, but that the remaining layers of Bruch's membrane are formed later, concurrently with the development of the choroid. It would thus appear that the outer portion of this membrane is formed by the choroid, while the inner is derived from the neural ectoderm of the optic cup. This double origin is borne out by the finer structure of the membrane, which Salzmann[3] divided into an outer, or elastic, layer and an inner, or cuticular, layer. Sattler[4] was the first to describe the network of fibers in the outer lamella, and Smirnow[5] demonstrated that these were elastic fibers, with some collagenous fibers interspersed. The inner lamella is entirely homogeneous and forms the greatest thickness of the combined membrane. Wolfrum, however, recognized a third layer, made up of fine collagenous fibers and lying between the inner and the outer lamella, while Verhoeff and Sisson[6] expressed the belief that this third layer of connective tissue lies immediately external to the elastic lamella. The inner lamella is the true lamina vitrea. Wolfrum did not feel that this is homogeneous, but thought that it has a finely fibrillar structure and is a common product of the protoplasmic processes of the pigment epithelium and the fine endings of the collagenous fibers. For this discussion the function of the membrane is just as important as is its structure. This membrane must be easily permeable, for the rods and cones are dependent for nutrition on the choriocapillaris, from which they are separated by the membrane. That it is also permeable to cellular elements was shown by Sattler and Wolfrum, who noted leukocytes traversing it, and by Hanssen,[7] who saw red blood cells penetrating into it in the neigh-

2. Wolfrum, M.: Beiträge zur Anatomie und Histologie der Aderhaut beim Menschen und bei den höheren Wirbeltieren, Arch. f. Ophth. **67**:307, 1908.

3. Salzmann, M.: Anatomie und Histologie des menschlichen Augapfels im Normalzustande, Vienna, Franz Deuticke, 1912, p. 63.

4. Sattler: Ueber den feineren Bau der Chorioidea des Menschen, nebst Beiträgen zur pathologischen und vergleichenden Anatomie der Aderhaut, Arch. f. Ophth. (pt. 2) **22**:1, 1876.

5. Smirnow: Zum Bau der Chorioidea des erwachsenen Menschen, Arch. f. Ophth. **47**:451, 1899.

6. Verhoeff, F. H., and Sisson, R. J.: Basophilic Staining of Bruch's Membrane, Arch. Ophth. **55**:125, 1926.

7. Hanssen: Zur Frage der Glashautdrusen der Aderhaut, Klin. Monatsbl. f. Augenh. **58**:249, 1917.

borhood of hemorrhages. Axenfeld [8] stated the belief that it is possible for choroidal elements to pass through the intact membrane.

Müller found that the earliest change in the senile type of drusen is thickening of the lamina vitrea, with consequent formation of elevations of varying form and height. Smaller drusen are covered with normal-appearing pigment epithelium, but over the larger ones the epithelial coating is flattened or even lacking. The drusen are of a hyaline appearance, though Kerschbaumer [9] showed that in their earliest stages they have a finely granulated phase. Alt [10] has demonstrated that the older nodules lose their homogeneous aspect and show concentric lamination. Later still, calcium may be deposited in them, and ultimately formation of bone may occur in the mass. Isolated nodules may attain large size, or by fusion of adjacent nodules structures of bizarre shape may be produced.

Many theories have been proposed for the origin of drusen of the lamina vitrea. Donders [11] felt that they result from degeneration of the nuclei of the pigment epithelium. De Vicentiis [12] thought that degeneration of the entire pigment cell is the original cause. Leber [13] regarded the hyalin as a secretion from the pigment cells. Alt [10] expressed the view that the cells produce the hyalin as a secretion and then become hyalinized themselves. It was da Gama Pinto's [14] opinion that the cells proliferate first and that the secretion forms in the center of each clump. Kerschbaumer [9] found that the lamina vitrea of elderly persons contained granules, which became fused and formed the nodules. He stained them with eosin, fuchsin and picrocarmine, and from the reactions of the granules and the nodules he concluded that they were a hyaline secretion from the pigment epithelium. Schieck [15] was of the opinion that the pigment epithelium desquamates some of its cells,

8. Axenfeld, T.: Retinitis externa exudativa mit Knochenbildung im sehfähigen Auge, Arch. f. Ophth. **90**:452, 1915.

9. Kerschbaumer, R.: Ueber Altersveränderungen der Uvea, Arch. f. Ophth. (pt. 1) **38**:127, 1892.

10. Alt, A.: Contributions to the Pathological Anatomy of the Human Eye, Arch. Ophth. & Otol. **6**:304, 1877.

11. Donders, F. C.: Beiträge zur pathologischen Anatomie des Auges, Arch. f. Ophth. (pt. 2) **1**:106, 1855.

12. de Vicentiis: Corpi vitrei della coroidea, Jahresb. ü. d. Leistung. d. Ophth. **5**:348, 1874.

13. Leber, T.: Ueber Retinitis pigmentosa und angeborene Amaurose, Arch. f. Ophth. (pt. 3) **15**:1, 1869.

14. da Gama Pinto: Anatomische Untersuchung eines nach Critchett's Methode wegen Hornhautstaphyloma operierten Auges, Arch. f. Ophth. (pt. 1) **28**:170, 1882.

15. Schieck: Zur Genese der sogenannten Drusen der Glaslamelle, Ber. ü. d. Versamml. d. ophth. Gesellsch., 1903, p. 320.

which degenerate and then become covered with pigment epithelium. Rumschewitsch [16] expressed his belief in the theory of a cuticular secretion but thought that later the cells themselves become hyalinized. Against these views that drusen derive from the pigment epithelium there are several others that seem unlikely. Rudnew [17] considered that leukocytes can wander into the epithelium and undergo degeneration to produce these bodies. Pes [18] offered the view that scars occur in the choroid following inflammation and by contraction produce papillary projections of the choroid, which become hyalinized. Hofmann [19] believed that drusen can result from the breaking through of hyaline thrombi from the choriocapillaris. Hanssen [7] also was of the opinion that the choroid produces the drusen, basing his belief on the fact that connective tissue elements are present in many of them.

Coats [20] carefully analyzed the chief theories and has divided them into two groups. The first theory, which he called the transformation theory, is based on the transformation of the pigment epithelium cells into the hyaline masses. According to this theory, the pigment epithelium undergoes senile changes whereby the layer of cells becomes irregular, some of the cells being larger than others. The pigment granules become irregularly distributed, and either colloid globules appear in the protoplasm or the entire cell becomes converted into a colloid body. It has been offered in favor of this theory that many of the colloid bodies show no connection with the lamina vitrea, which appears to pass unchanged beneath them. Coats, however, was opposed to this view. He did not feel that the transition of the pigment cells into drusen has been sufficiently demonstrated. Also important to him was the objection that the nodules remain covered with pigment epithelium, even when they have attained considerable size. This convinced him that the excrescences originate beneath the epithelium and have elevated it, for he could not agree with the idea that the epithelium would proliferate and cover the nodules.

The second, or deposition, theory was more to his liking, and he presented a strong case in its favor. First he offered the analogy of

16. Rumschewitsch, K.: Zur pathologischen Anatomie der sogenannten Drusen der Glaslamelle der Aderhaut, Klin. Monatsbl. f. Augenh. **42**:358, 1904.

17. Rudnew: Ueber die Entstehung der sog. Glaskörper der Choroidea des menschlichen Auges und über die hyaline Degeneration der Gefässe derselben, Arch. f. Ophth. **53**:455, 1871.

18. Pes, O.: Die glasigen Körper und Papillarbildungen der Chorioidea, Arch. f. Ophth. **59**:472, 1904.

19. Hofmann: Ein Beitrag zur Kenntnis der Gefässveränderungen im Auge bei chronischer Nephritis, Arch. f. Augenh. **44**:339, 1902.

20. Coats, G.: The Structure of the Membrane of Bruch, and Its Relation to the Formation of Colloid Excrescences, Roy. London Ophth. Hosp. Rep. **16**:164, 1904-1905.

the excrescences on Descemet's membrane and the capsule of the lens, which he felt are definitely produced by the endothelial cells on the posterior corneal surface and by the cells of the lenticular epithelium, respectively. He then discussed the critical point that in many sections it has been noticed that Bruch's membrane passes beneath the drusen apparently intact. He felt that in some cases this is due to sectioning the periphery of the excrescence and thus not noticing the stalk which is connected to the membrane. However, he was convinced that the real explanation lies in the structure of Bruch's membrane, for the lamina elastica does not partake in this condition and passes unimpaired beneath the excrescences. When stained with eosin this portion seems to be the entire membrane, but when Weigert's elastic tissue stain is used it will be seen that only the outer portion of the lamina elastica is intact. The excrescence will be found to be a true bulging of the homogeneous lamina vitrea and, like it, will not stain specifically with Weigert's stain. Though Coat's conclusions are in favor of the deposition theory, he did state that both theories assume an abnormal metabolism of the pigment epithelium. He made no attempt to give the cause of this metabolic disturbance but felt that at times it is definitely pathologic, as evidenced by the occurrence of colloid bodies in diseased eyes. At other times, however, he regarded it as almost a normal change, since it is present so commonly in senile eyes.

It is my purpose in this study to present those transitional stages in the pigment epithelium which Coats felt are a weak link in the chain of argumentation in favor of the transformation theory. This evidence has been noted only in eyes which have suffered degeneration from injury or inflammation, and not in the group of senile eyes. Unquestionably the pigment epithelium during senility is subject to metabolic disturbances, and it is probably able to produce excessive amounts of the hyaline secretion, which deposits on the lamina vitrea in the form of excrescences. Consequently I shall not venture to disagree with Coats and the other proponents of the deposition theory with regard to the senile type of drusen. However, I do not feel that conditions are the same in degenerating eyes. The pigment epithelium in such eyes is subjected to a much greater noxious influence and reacts differently to produce a different type of excrescence. This does not mean that both types of drusen cannot occur in the same eye, for many eyes which contain the senile type of drusen can undergo subsequent degenerative changes.

The pigment epithelium of the retina can apparently suffer changes in its metabolism as a result of either choroidal or retinal disturbances. This is understandable when it is realized that this layer is subjected

to the cross-current of the metabolic products of the retina and the nutrient substances from the blood stream in the choriocapillaris. The earliest manifestation of the decline of this layer is the irregularity of the cells, which under normal conditions are uniformly arranged.

Fig. 1.—Swelling of isolated cells of the pigment epithelium; × 1,050.

Fig. 2.—Protrusion and beginning hyalinization of cells of the pigment epithelium; × 1,050.

Figure 1 shows isolated cells becoming swollen and pushing themselves out above the layer, though maintaining their basis on the lamina vitrea. The protoplasmic mass becomes distended, and with the dispersion of the granules of pigment the nucleus is more distinctly visible. Figure 2 illustrates the next stage, in which the cells project still farther

and are beginning to undergo hyalinization. Here it will be seen that the nucleus is becoming indistinct and that the protoplasm begins to show numerous droplets. In a case of disklike degeneration of the macula Holloway and Verhoeff[21] saw in one eye collections of swollen transparent cells filled with fine granules, which were pigmented cells that had undergone fatty degeneration, and all the transition stages in the process could be traced. In the other eye the pigment epithelium showed marked degenerative changes, with formation of numerous colloid excrescences. Along one stretch the cytoplasm and nuclei had completely disappeared, and there was a layer of colloid balls, over which there remained granules of pigment left by the pigment cells.

The response of the pigment epithelium and its subsequent changes are probably dependent on the type of baneful agent and the amount

Fig. 3.—Proliferation of the pigment epithelium, with hyalinization in the center of the clump; × 850.

of damage to the cells. These cells have the capacity of rapid proliferation and in such cases will form clumps, which may be seen protruding from the surface, as in figure 3. Since these are not healthy cells, they quickly undergo degenerative changes, and the masses are composed of a surrounding capsule of new cells with an inner core of degenerated and hyalinized older ones. The drusen can increase in size considerably in such a manner, for as the older cells degenerate new ones proliferate, increasing the bulk of the mass. This manner of growth will also account for the layer-like structure of some of the nodules, which is illustrated in figures 4 and 5. In these two figures it can also be seen that the process is not a uniform one, for some cells

21. Holloway, T. B., and Verhoeff, F. H.: Disc-Like Degeneration of the Macula, Tr. Am. Ophth. Soc. **26**:206, 1928.

incarcerated in the masses have not as yet undergone complete loss of their structure. It must be borne in mind, furthermore, that the pigment epithelium retains its secretory powers, even though its position has been changed, and that the rapidly proliferating cells are probably adding their hyaline secretion to the conglomerate. This peripheral

Fig. 4.—Hyalinized masses showing some incarcerated cells which have not yet completely degenerated; × 420.

Fig. 5.—Layers of hyaline material showing some connection to the lamina vitrea, and interspersed layer cells of proliferated pigment epithelium; × 300.

accretion is most likely the cause of some of these drusen having a concentrically layered structure, as is shown in figure 6 and, under higher magnification, in figure 7.

When the pigment cells are more severely damaged they do not have the power to proliferate, but along the entire layer there will be areas

Fig. 6.—Laminated masses of hyalin completely encapsulated by proliferated pigment epithelium; × 160.

Fig. 7.—High power magnification of section of figure 6, showing details of lamination; × 915.

of lipoidal degeneration of the cells. Figure 8 shows this foamy granular mass beneath the fragmented and disarranged epithelium. Such masses have been reported in animals fed high cholesterol diets and have been cited by Heath [22] and by J. Friedenwald,[23] with the suggestion that disturbances of the lipoid metabolism are a possible cause of the formation of drusen.

Many drusen contain connective tissue elements and areas of calcification and formation of bone. From this Hanssen has assumed that the choroid alone is entirely responsible for the formation of the drusen. It is not necessary to assume this, for with inflammatory disturbances present in the choroid the lamina vitrea can be broken through and connective tissue thus invade the drusen, leading to their organization. Figure 9 shows such an area of calcification beginning in the region of the choroid and extending up into the mass. Also Holloway and Verhoeff [21] have demonstrated that connective tissue cells can be

Fig. 8.—Extensive lipoidal degeneration of the pigment epithelium forming deposits on the lamina vitrea; × 800.

derived from the pigment epithelium cells themselves, and this probably explains the frequency of the presence of these elements. Nevertheless, these authors stated that they have seen ossification of a large colloid excrescence produced by connective tissue which had entered it from the choroid.

That these colloid masses are different from the homogeneous nodules found in the senile eyes is without a doubt. As can be seen in figure 10, the senile variety of drusen shows none of the bizarre structure seen in the degenerative type, for here one has smooth homogeneous nodules encapsulated by flattened, pigmented epithelial cells and corresponding in all details to their ophthalmoscopic appearance. Granting Coat's thesis that the only valid explanation for their origin

22. Heath, P.: Ocular Lipid Histiocytosis and Allied Storage Phenomena. Arch. Ophth. **10**:342 (Sept.) 1933.

23. Friedenwald, J.: The Retinal Blood Vessels in Arteriosclerosis and Hypertension, Folia ophth. orient. **2**:175, 1936.

is the deposition theory, I do not feel that this theory can be applied to the entirely different structures seen in degenerating eyes. Many observers have attempted to differentiate the two varieties of drusen by staining methods, but these do not offer any definite evidence. It
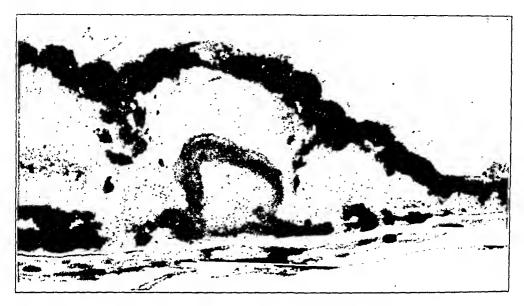
Fig. 9.—Nodule of calcification in a hyaline mass; × 1,000.
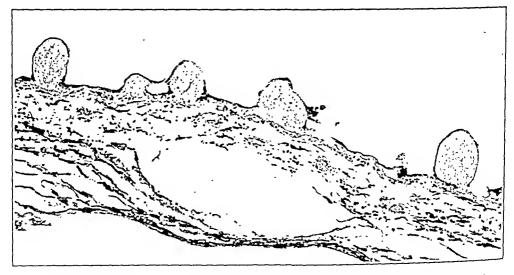
Fig. 10.—Senile type of drusen showing smooth hyaline nodules attached to the lamina vitrea and encapsulated by flattened pigment epithelium; × 205.

has been noted that with the Van·Gieson stain the senile variety is yellow, while the other variety stains red. Lauber [24] has stained a

24. Lauber: The Origin of Hyaline Formations Within the Eye, Ber. ü. d. deutseh. ophth. Gesellseh. **44**:216, 1924.

number of phthisical eyes for fat. He found that where the deposits between the lamina vitrea and the pigment epithelium were stained bright red with the sudan stain, red granules could be recognized in the neighboring pigment epithelium, which he assumed to mean the presence of a chemically similar substance both in the pigment epithelium and in the deposits. I have attempted to apply the Molisch reaction for carbohydrates as a microchemical test, in order to determine whether these structures would react in the same way as Bruch's membrane, but was unsuccessful in applying this method to microscopic preparations.

Schreiber [25] has approached the problem from an experimental angle. He was indebted to Wagenmann's investigations for the knowledge that as a result of partially severing the short ciliary arteries in dogs there is a disturbance of the choroidal circulation followed by a quick degeneration of all the retinal layers, especially the rods and cones. In the neighborhood of the circulatory disturbance the pigment epithelium showed marked proliferation, with penetration of the pigment cells into the atrophic retina. Schreiber repeated these experiments and observed that numerous pigment cells had wandered completely through the retina and formed on its inner side irregular masses of cells, but that in some places they had arranged themselves in a single layer of epithelium. However, he was particularly interested in the appearance of numerous drusen-like structures. These were present not only where the epithelium lies on the lamina vitrea but also at the places to which it has migrated. Thus, they were seen on the inner surface of the retina, and because of their relationship to the pigment epithelium and their concentrically layered appearance their character could not be doubted.

In conclusion, it would therefore appear that enough evidence has been accumulated to controvert Coat's objections to the transformation theory of the formation of drusen, at least as to the formation of those drusen occurring in degenerating eyes. The transitional stages from pigment epithelium to drusen are well shown by the illustrations. The other objection—that the nodules remain covered with pigment epithelium and consequently must arise beneath it—is well answered by Schreiber's work, for he has demonstrated that, with the migration of the pigment cells into the retina and their subsequent degeneration, colloid masses are formed and are either completely or partially surrounded by pigment cells.

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25. Schreiber, L.: Ueber Drusenbildung des Pigmentepithels nach experimenteller Ciliararterien-Durchschneidung beim Kanninchen, Ber. u. d. Versamml. d. ophth. Gesellsch., 1906, p. 286.

ABSTRACT OF DISCUSSION

Dr. Frederick H. Verhoeff, Boston: Dr. Rones has given an excellent account of the various theories that have been advanced as to the histogenesis of drusen and has utilized specimens of his own to illustrate certain important histologic facts relating to these peculiar bodies. For convenience he classifies drusen into two main types, the senile and the degenerative, admitting, however, that each type can occur in senile eyes and also in degenerating eyes. In the senile type the substance of the body is homogeneous and hyaline in appearance, while in the degenerative type the body contains, in addition to hyaline substance, products of cellular disintegration and material such as granules and droplets of lipoid.

Dr. Rones concludes that the degenerative type results from degenerative transformation of the cells, although he assumes, inconsistently, it seems to me, that some of the hyalin is secreted by the cells concerned. He contends that his observations controvert Coats' objections to the theory of transformation. In my opinion, however, they do not throw any doubt on the validity of Coats' theory or indicate that this theory is not applicable to the degenerative type of drusen. Coats held that the inner layer of Bruch's membrane consists of a hyaline substance originally secreted by the cells of the pigment epithelium and that drusen result when certain cells later secrete an additional amount of the same substance. The question as to whether or not the hyaline substance of drusen is exactly similar to that of the cuticular layer of Bruch's membrane seems unimportant to me, since under abnormal conditions they might well differ somewhat in composition. The fact that they are both hyalin-like is sufficient. Also, it seems unimportant whether or not they are demarcated from each other.

The important question is whether or not the hyaline substance accumulates within the cells or is deposited beneath them. My own observations indicate that it is deposited beneath the cells. If it accumulates first within the cells, it should be possible at some stages to identify it there, but I have never been able to do so. The concentric lamination often seen in the larger drusen seems to me more consistent with deposition of secretion in layers than with transformation of degenerating cells.

Dr. Rones' observations do not exclude the possibility that the hyaline material found in drusen of the degenerative type is not a secretion of the cells. If it is a secretion Dr. Rones' observations do not in any way controvert Coats' theory. According to my conception, the simplest type of excrescence is due to abnormal activity of a single cell and the secretion by the cell of hyaline or colloid material beneath it which gradually pushes the cell above the surrounding surface. The body then becomes larger because secretion is added to it by the surrounding cells. When it becomes of considerable size or, in some cases, when it is still quite small, lime salts, fat and sometimes cholesterol, may be deposited within it, just as in the case of many inert substances.

Under more severe degenerative conditions there are irregular proliferation and necrosis of cells and irregular formation of hyaline substance which sometimes precede and sometimes follow the severe degenerative process. In this way the various bizarre appearances referred to by Dr. Rones result.

Under certain conditions cells of the pigment layer can assume the appearance of fibroblasts and lay down a substance that cannot be distinguished from hyaline connective tissue. I believe this is similar to the substance that the pigment cells produce when they form drusen. Often a new-formed layer of pigment cells produces beneath it a cuticular membrane resembling that of Bruch's membrane.

I shall show sections of two drusen of unusual size, which illustrate some of the facts to which I have referred and also show organization of drusen partly by proliferating pigment epithelium and partly by mesoblastic cells and blood vessels derived from the choroid. They also show calcification of the drusen.

I shall demonstrate on the blackboard the relation to drusen of the fenestrated membrane in the pigment layer that I described many years ago but which still often passes unrecognized. I later found that this membrane had been previously described but not interpreted as a membrane. It is as truly a membrane as the external limiting membrane of the retina, with which it can sometimes be seen continuous at the margin of the disk.

Attention has often been called to the similarity of drusen to the so-called warts on Descemet's membrane, but I do not recall that any one has hitherto pointed to an analogy between them and the hyaline formations often to be seen in the thickened corneal epithelium in cases of bullous keratitis. I shall show a lantern slide which renders this analogy obvious. In some cases there is even produced a duplication of Bowman's membrane. Probably Bowman's membrane and the cuticular layer of Bruch's membrane are but basement membranes such as commonly occur beneath epithelium everywhere.

An interesting question is whether drusen in otherwise normal eyes are due to so-called abiotrophy or to some obscure local or general disturbance of metabolism. Certainly, as is noted by Dr. Rones, they may occur in young persons. I recall seeing them with the ophthalmoscope abundant in the fundi of a girl 15 years of age.

The pigment epithelium from many points of view has been insufficiently investigated, and I am sure that Dr. Rones' communication will stimulate interest in the study of this important structure.

Dr. Arthur J. Bedell, Albany, N. Y.: After the excellent presentation by Dr. Rones and the enlightening discussion by Dr. Verhoeff, further remarks on the pathologic problems associated with drusen seem unnecessary. I shall confine my discussion and demonstration to the thought suggested in the first paragraph of Dr. Rones' paper, the ophthalmoscopic appearance of drusen.

The fundic picture depends on the general conformation of the background, the size and number of the deposits and the degree of pigmentation.

Isolated drusen are bright yellow dots, which always lie beneath the retinal vessels. When numerous or confluent they are whiter and may form a definite layer parts of which may be thin and other portions so thick as to be almost nodular.

In cases of extensive involvement a wide zone of white spots almost completely surrounds the disk but usually avoids the region of the macula. In the cases in which the drusen are least developed the spots are discrete and more often distributed about the macula, and

occasionally they are small, brilliant and close to the nasal side of the disk. They always show a measurable degree of elevation, which is readily determined by parallactic motion or slit lamp inspection.

Macular disease may develop in an eye in which drusen are present. This confuses the picture and may lead to delay and error in diagnosis. When drusen are isolated they never cause any visual loss, and even when massed in the macular area they rarely interfere with the visual function.

There is little difficulty in making the correct diagnosis in cases in which the condition is simple and uncomplicated, but in those in which there are more intricate patterns it is well to remember that the drusen may simulate the retinal exudate found in some of the chronic inflammatory diseases of the fundus. A sheet of drusen may be mistaken for the white dots that form in the retina after the subsidence of papilledema. Careful scrutiny will, however, prove that the latter deposits are dull white and sometimes even overlie a contiguous vessel.

The white spots of so-called retinitis punctata albescens are present only when there are other evidences of retinal disease, such as peripheral pigmentation, narrow retinal vessels or, in cases in which the condition is advanced, atrophy of the optic nerve associated with pallor of the disk.

When signs of degeneration of the fundus are present and recognized, differentiation between drusen and other pathologic states is easy.

When drusen are first visible with the opthalmoscope they are small spots slightly pinker than the surrounding fundus. As they enlarge they become more yellow; then they grow paler, showing some pigmentation. The larger aggregations are often white, and not infrequently the lesion has a gray or dark brown outlining ring.

DR. BENJAMIN RONES, Washington, D. C.: With respect to Dr. Bedell's beautiful photographs, I wish to call to attention that these are not the kind of drusen that I was particularly interested in, for these are the senile variety.

Dr. Verhoeff believes that the pigment epithelium has the power to secrete the hyaline substance. This is assumed by most ophthalmologists. However, it is difficult to conceive that with such a marked disturbance in the cell itself these damaged cells have the power to produce large amounts of secretion.

It is easier to conceive that the cell itself is undergoing necrosis and hyalinization, and I attempted in these photographs to show that these damaged cells would become conglomerate and contain the pigment within them and form these layered structures.

Dr. Verhoeff has shown a layered structure of a somewhat similar nature in the corneal epithelium. However, in looking at that slide one is struck by the fact that the included cells are perfectly healthy, and the surrounding epithelium is apparently healthy, as contrasted with the pigment epithelium in these other sections, which were undoubtedly considerably damaged.

SEASONAL VARIATIONS IN THE LIPID CONTENT OF THE CRYSTALLINE LENS

P. W. SALIT, Ph.D.

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A general survey of a series of biochemical studies of human cataractous and sclerosed lenses [1] revealed distinct variations in the total lipid content at different times of the year. Such variations coincided with the four seasons. On the average, the total lipid content was low in the spring and autumn and high in the summer and winter.

Analyses of 128 lenses for determination of the total lipid content were carried out over a period of fourteen months, i.e., from March 1935 to April 1936, inclusive.

The patients from whom the lenses were extracted came from different parts of Iowa, and the operations were performed at such intervals that not a single week passed without a few lenses being analyzed. The smallest number of lenses analyzed during the course of one month (April 1935) was 11; the largest number was 23 (May and November 1935; table 1). Owing to this rather uniform distribution of material throughout the course of the study, it was possible to obtain not only monthly, but also biweekly, averages. The values are plotted in chart 1 under c and b, respectively.

Each cataract was studied and its stage of maturity determined with the biomicroscope, and later it was removed by the intracapsular method. Of the 128 lenses, 79 (61.7 per cent) had incipient cataract, 12 (9.4 per cent) intumescent cataract and 37 (28.9 per cent) mature cataract. Incipient cataract, in its biochemical make-up, is practically like the normal lens; intumescent cataract and mature cataract differ from the normal lens chiefly with respect to their water content, this being higher in the two former. There is also a slight increase in the amount of total lipids in lenses with advanced cataract and those with sclerosis. In this series of cases each monthly accumulation of lenses is represented by the three types of cataracts, i.e., incipient, intumescent and mature, in approximately the same proportion. This applies naturally also to their sclerotic conditions. There is likewise a tendency for the amount of total lipids to increase with age, which in this case varied between

41 and 87 years. This increase was greater on the basis of absolute amounts, owing chiefly to the larger size of the older lenses, but on the basis of percentage it amounted, on an average, to less than 0.3 per year and is negligible as to its influence on the character of the curve. Moreover, the average ages of the patients from which the lenses were obtained during the course of each month did not vary considerably, and when plotted against months showed practically no correlation to the curve for the lipid content. Therefore, the variations in the lipid content at different times of the year could not be due to factors other than seasonal influences.
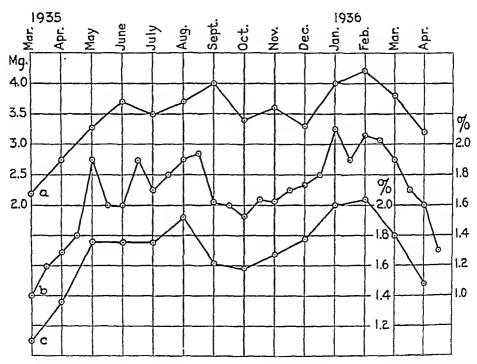
Chart 1.—Average values for the total lipid content. *a* shows the values in terms of absolute amount, or milligrams of total lipids per lens; *b* shows the biweekly values in terms of percentage, and *c*, the monthly values in terms of percentage.

Each lens was analyzed separately by a micromethod described in a previous publication.[1] In this method the lipids were extracted by solvents of fat, such as chloroform, alcohol, ether and acetone. After the evaporation of the extractives at a temperature of 65 C., the amount of lipids was estimated gravimetrically with a sensitive chemical balance.

The absolute values for lipids for the entire period of fourteen months varied between 1.2 and 6.1 mg. per lens. The values in terms of percentage varied between 0.897 and 3.142. The weights of the individual lenses varied between 0.1343 and 0.3699 Gm. The average weights of the lenses for each month, however, differed only slightly, and when plotted against months, appears to be of no significance.

The absolute value for lipids varied somewhat with the seasons of the year (table 1 and chart 1, *a*): they were lowest in the spring, increased somewhat during the summer, then decreased slightly during the fall and reached their maximum in the winter. On the basis of percentage, the seasonal variations were much more pronounced (table 1, and chart 1, *b* and *c*). The average value for March 1935 was 1.10 per cent (chart 1, *c*). There was then a steady increase throughout the succeeding months until a value of 1.92 per cent was reached in August. This means an increase of nearly 73 per cent over the value for March. Then the value decreased, reaching a relatively low level of 1.59 per

TABLE 1.—*Seasonal Variations in the Total Lipid Content of Human Crystalline Lenses*

Month	Number of Lenses	Average Age of Patients, Years	Average Weight of Lenses, Gm.	Average Amount of Total Lipids per Lens, Mg.	Average Amount of Total Lipids, Percentage
1935					
March	15	63.6	0.203	2.3	1.10
April	11	69.7	0.194	2.8	1.35
May	23	70.7	0.193	3.3	1.75
June	19	73.2	0.209	3.7	1.75
July	12	63.6	0.193	3.5	1.75
August	14	69.6	0.205	3.7	1.92
September	13	67.8	0.210	4.0	1.62
October	15	69.2	0.213	3.4	1.59
November	23	69.6	0.214	3.6	1.67
December	15	61.3	0.189	3.3	1.77
1936					
January	21	63.7	0.192	4.0	2.00
February	13	68.3	0.209	4.2	2.04
March	17	67.5	0.208	3.7	1.80
April	17	69.2	0.212	3.2	1.47

cent in October—a level indicating a drop of nearly 21 per cent as compared with the value for August but more than 43 per cent higher than that for March of the same year. A second high peak, of 2 per cent, was reached in January 1936, and a peak of 2.04 per cent was reached in February 1936, which is almost double the value for March of the preceding year and more than 26 per cent higher than that for October 1935. The value then decreased rather rapidly, until by April 1936 it reached 1.47 per cent, which is more than 8 per cent lower than the value for October of the previous year.

The graph based on monthly averages (chart 1, *c*) has a rather smooth curve, showing that these variations are not merely chance but are the result of profound systematic and periodic changes in physiologic processes.

Previous to the analysis of the total lipid content in the crystalline lenses, similar analyses had been carried out for determination of the cholesterol and phospholipid contents.[2] The cholesterol content was determined by the colorimetric method of Bloor.[3] For the estimation of the phospholipid content a modification of Whitehorn's [4] colorimetric method was employed. The ages of the patients whose lenses were analyzed varied between 45 and 85 years in the cases in which the cholesterol content was determined and between 41 and 87 years in the cases in which the phospholipid content was determined. Unfortunately, the number of lenses analyzed each month was very small in both series of cases, not quite half that in the cases in which the total lipid content was determined, but the analysis for the cholesterol content extended
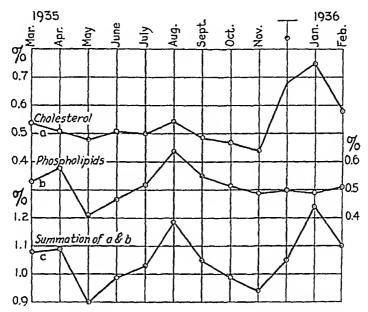
Chart 2.—Average values for the cholesterol and phospholipid contents in terms of percentage.

over fifteen months and that for the phospholipid content over sixteen months, as compared with fourteen months for the determination of the total lipid content. In order to obtain more reliable averages the differ-

2. (a) Salit, P. W., and O'Brien, C. S.: Cholesterol Content of Cataractous Human Lenses, Arch. Ophth. 13:227-237, 1935. (b) Salit, P. W.: Phospholipid Content of Cataractous Human Lenses, Brit. J. Ophth. 19:663-671, 1935; (c) Phospholipid Content of Cataractous and Sclerosed Human Lenses: A Biochemical Study of Lenticular Changes, Arch. Ophth. 16:271-283 (Aug.) 1936.

3. Bloor, W. R.: Studies on Blood Fat: II. Fat Absorption and the Blood Lipoids, J. Biol. Chem. 23:317-326, 1915; The Determination of Small Amounts of Lipid in Blood Plasma, ibid. 77:53-73, 1928.

4. Whitehorn, J. C.: A Method for the Determination of Lipoid Phosphorus in Blood and Plasma. J. Biol. Chem. 62:133-138. 1924. Salit.[2c]

ent values for identical months were pooled, and curves were obtained
for only one year, i. e., twelve months. This was first done for the
values for cholesterol and the phospholipids separately (chart 2, *a* and
b, and chart 3, *a* and *b*). A third curve was obtained by the addition
of these two (chart 2, *c,* and chart 3, *c*). On account of the small num-
ber of lenses, no great accuracy can be claimed for these curves. They
do, however, bear the character of certain definite periodic variations
or tendencies. The influence of the factor of age was operative here
even less than in the case of the total lipid content, although there are
two high points in each curve coinciding with rather high averages for
age; the influences of the latter may account for only an exceedingly
small part of the high values. Similarly, the fact that some of the lenses
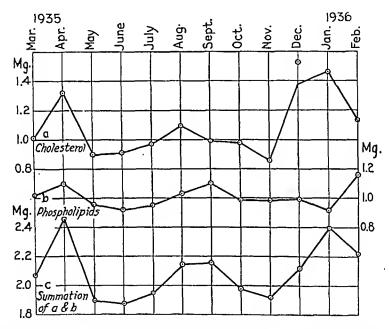
Chart 3.—Average values for the cholesterol and phospholipid contents in
terms of absolute amount, or milligrams per lens.

had a mature cataract has no bearing on the nature of the curves for
the same reason that was pointed out in connection with the total lipid
content. The variations, therefore, as in the case of the total lipid con-
tent, must be attributed to seasonal influences alone. In the curves show-
ing the values in percentage (chart 2, *a* and *b*) both the cholesterol
content and the phospholipid content are seen to be low in May. They
increased during the early months of summer, reaching a high level
in August; this increase was especially pronounced in the phospholipid
content. This was followed in both cases by another decline, the lowest
level being reached in November. In December only 1 lens was analyzed
for the cholesterol content, and it happened to be the one with the highest
cholesterol content. As a single value this content has no significance,

but in conjunction with the cholesterol content of the 3 lenses analyzed in January, which likewise happened to be of a relatively high cholesterol content, it indicates a second and a more pronounced increase in the cholesterol content after the onset of winter, the highest level being

TABLE 2.—*Seasonal Variations in the Cholesterol Content of Human Crystalline Lenses*

Month	Number of Lenses	Average Age of Patients, Years	Average Weight of Lenses, Gm.	Average Amount of Cholesterol per Lens, Gm.	Average Amount of Cholesterol, Percentage
March	6	66.0	0.201	1.04	0.54
April	10	63.6	0.196	1.34	0.50
May	9	66.1	0.195	0.93	0.48
June	3	69.0	0.189	0.93	0.51
July	6	70.1	0.205	0.99	0.50
August	16	65.6	0.206	1.11	0.54
September	18	70.7	0.222	1.04	0.48
October	18	62.4	0.215	0.99	0.46
November	9	64.1	0.204	0.89	0.44
December	1	79.0	0.186	1.54	0.83
January	3	74.0	0.207	1.47	0.74
February	6	61.8	0.203	1.15	0.58

TABLE 3.—*Seasonal Variations in the Phospholipid Content of Human Crystalline Lenses*

Month	Number of Lenses	Average Age of Patients, Years	Average Weight of Lenses, Gm.	Average Amount of Phospholipid per Lens, Mg.	Average Amount of Phospholipid, Percentage
March	5	60.0	0.197	1.04	0.53
April	9	70.9	0.209	1.12	0.58
May	6	69.2	0.204	0.97	0.41
June	3	68.3	0.205	0.94	0.47
July	3	62.0	0.196	0.96	0.52
August	2	74.0	0.163	1.04	0.64
September	12	67.0	0.206	1.10	0.55
October	7	69.5	0.193	0.99	0.52
November	16	69.6	0.195	0.95	0.49
December	12	68.8	0.203	1.01	0.51
January	9	68.2	0.192	0.92	0.49
February	17	63.8	0.220	1.08	0.50

reached during December and January. A lower cholesterol content was registered during February, yet this value was still considerably higher than any of the values obtained during the spring, summer and fall. On the other hand, there were practically no further changes in the phospholipid content after November throughout the winter.

By summation of the two curves a third curve (chart 2, *c*) was obtained, which strikingly resembles the one for the total lipid content; this shows that the low levels were in May and November, and the high points coincide with August and January.

When the corresponding absolute values for cholesterol and the phospholipids are plotted against the twelve months of the year (chart 3, *a* and *b*), they resemble the ones based on percentage. The seasonal variations in the phospholipid content, however, are shown to be not as pronounced as those obtained on the basis of percentage, but the variations in the absolute amounts in the curve for cholesterol are seen to be even more definite than those in the curve obtained on the basis of percentage. By summation of the curves for absolute amounts of cholesterol and that for absolute amounts of phospholipids, a third curve (chart 3, *c*) was obtained which resembles closely the summated curve obtained on the basis of percentage and consequently also the curve for the total lipid content.

The total lipids of the crystalline lenses consist of approximately equal amounts of cholesterol, phospholipids and ordinary fat.

From the foregoing statements it seems certain that the lipid content of crystalline human lenses is subject to seasonal variations; these variations are chiefly due to changes in the cholesterol and the phospholipid content.

SUMMARY

A total of 128 human crystalline lenses were analyzed for the total lipid content over a period of fourteen consecutive months. The lenses, which consisted of 79 with incipient cataract, 12 with intumescent cataract and 37 with mature cataract, were removed from the eye in toto by the intracapsular method. The lipids of each lens were extracted separately by the use of such solvents of fat as chloroform, alcohol, ether and acetone, and their amounts were estimated gravimetrically with a sensitive chemical balance.

By plotting the monthly averages of lipids, both in terms of percentage and in terms of absolute amount, a characteristic, four-phasic curve was obtained, in which the two crests coincide with summer and winter and the two troughs coincide with spring and fall. Since nearly 62 per cent of the lenses had incipient cataract or were practically normal and the differences in the composition of the lipids of mature cataract as compared with normal lenses consist only in a slightly increased total lipid content and a reduced phospholipid content, these striking variations as represented by the curve must be attributed solely to seasonal influences.

Two groups of similar lenses had been previously analyzed for the cholesterol content and the phospholipid content, respectively. By plot-

ting their values in the same way, curves were obtained of a more or less diphasic nature but of opposite tendencies; the curve for the phospholipid content shows a pronounced increase in the content during the summer and a relatively low value during the rest of the year, whereas the curve for cholesterol content has its highest elevation during the winter and its lowest levels during the rest of the year. When the two curves were summated a third curve was obtained which strikingly resembles that for the total lipid content, the level for the phospholipid content representing the elevated part during the summer, that for the cholesterol content representing the elevated part during the winter and the low levels coinciding with the spring and autumn, exactly as in the curve for the total lipid content.

ABNORMAL OCULAR AND PUPILLARY MOVEMENTS FOLLOWING OCULOMOTOR PARALYSIS

REPORT OF A CASE

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On recovering from palsy of the oculomotor nerve, the formerly paralyzed ocular muscles may make movements which are contrary to or incoordinate with normal associated motions of the globes and eyelids. For example, in looking down, the upper eyelid, which normally follows the globe in its vertical movements, may not descend. Indeed, the eyelid may even retract upward when the globe moves downward. Another atypical ocular movement abnormally associated with downward gaze is inward rotation of the globe. A third and more rare type of abnormal movement associated with looking down is constriction of a pupil which at rest is dilated and inactive to light or in accommodation. In the following case report all three types of abnormal movements occurring simultaneously in one eye are described.

REPORT OF CASE

J. D., a man aged 28, applied to the dispensary of the Lincoln Hospital for treatment of a contused lip. On examination it was noted that when the patient looked down the left upper eyelid moved upward, the eyeball moved slightly inward, and the pupil constricted. When questioned as to his ocular condition, the patient replied that he was treated in the Bellevue Hospital in 1930 for headache and. closure of the left eye.

The history recorded in the Bellevue Hospital is that on July 2, 1930, while running through a rain storm, he felt a sudden pain in the nape of the neck. When he reached home three minutes later, he collapsed and became semiconscious. Later in the afternoon he became nauseated. In the evening he complained of severe occipital headache. On the following morning the headache settled over the left eyebrow. For the next few days, in addition to headache and drowsiness, there was vomiting. On July 8 it was noted that the left eyelid began to droop. Two days later the drooping of the lid became complete; he then entered the hospital.

Examination revealed the patient to be slightly stuporous, although cooperative. The significant findings were oculomotor ophthalmoplegia in the left eye with complete ptosis, external strabismus, paralysis of mesial and vertical movements of the globe, and a dilated pupil, which was fixed to light and in accommodation. The fundi were normal. There was a questionably positive Babinski sign on the right. The rest of the examination, including a complete test of the neurologic status, gave negative results.

Throughout the patient's stay in the hospital his temperature was normal. The pulse rate was slow, ranging between 54 and 64 beats per minute. The blood pressure was 120 mm. of mercury systolic and 65 mm. diastolic. Lumbar puncture performed on the day after admission revealed xanthochromic fluid which contained many crenated red blood cells. A second puncture, made three days later, showed bloody spinal fluid with supernatant xanthochromic fluid. The Wassermann tests of the blood and spinal fluid were negative. Cultures of the spinal fluid gave negative results. The blood count showed 9,100 white blood cells, with 72 per cent polymorphonuclear cells, 26 per cent lymphocytes and 2 per cent eosinophils. The urine was normal; there was no sugar or albumin.

Under observation the patient was drowsy, and he vomited on several occasions. There was slight and gradual improvement in his condition. At the end of two weeks' residence he felt well enough to leave the hospital against advice. The clinical diagnosis was subarachnoid hemorrhage due to a ruptured aneurysm. The subsequent history, as obtained from the patient, was that six weeks after he left the hospital the drooping eyelid began to open and since then he has had double vision.

Examination made at the dispensary of the Lincoln Hospital on Feb. 13, 1937, revealed oculomotor ophthalmoparesis, with abnormal movements of the eyelid, globe and iris of the left eye. When the patient was asked to look down the following phenomena were noted to occur in the left eye: (*a*) The superior eyelid retracted slightly upward. (*b*) The eyeball rotated slightly inward. (*c*) The pupil, which was 6.5 mm. in diameter and inactive to stimulation by direct or indirect light or in accommodation, contracted to 3 mm. The constriction of the pupil was slow and appeared one or two seconds after the patient began to look down.

Inward motions of the left eyeball as part of conjugate movements of the eyes to the right also produced retraction of the upper eyelid. No motion was noted in the upper eyelid or in the iris when the eyes gazed upward; the only abnormal movement noted in this position was slight internal rotation of the globe. Apparent ptosis of the left eyelid was present when the patient looked up. Vertical movements of the left eyeball were much impaired and more so for upward than for downward motion. The power of convergence was lost. Conjugate movements to the left were normal.

The patient was able to shut both eyes. During this procedure the right eyeball turned upward, whereas the left eyeball turned outward and slightly upward. Associated with the closure of the lids there was a slight constriction of the pupil in each eye. The corneal reflexes were normal.

When the patient was asked to look down, with the eyes initially closed, the left superior eyelid snapped upward.

All the ocular movements and reflexes in the right eye were normal. The optic fundi were normal. The visual fields when tested grossly showed no defects in any of the quadrants.

The deep tendon reflexes were slightly increased on the right. There was no Babinski sign. The gait, coordination, muscular power, and sensation, and the function of the cranial nerves other than that of those supplying the left eye were normal. There were no abnormal movements of the extremities, no nystagmus and no gross mental disturbances. Clinical examination of the thoracic and abdominal viscera gave negative results.

COMMENT

The simultaneous occurrence of retraction of the upper eyelid, inward rotation of the globe and constriction of the pupil in a paretic eye is not a frequent observation. The delayed and slow constrictions of an otherwise immobile pupil, in association with downward gaze or, less conspicuously, in association with closing of the eyes, are rare phenomena.

The failure of the superior eyelid in a formerly ophthalmoplegic eye to droop when the gaze is directed downward has been known as the pseudo-Graefe phenomenon.[1] This, however, is a misleading eponym which has crept into the ophthalmologic literature because the mechanism of the phenomenon was not clearly understood. There is no relation in mechanism or etiology between the lag of the lid in cases of exophthalmic goiter and that seen after recovery from oculomotor palsy. In the latter instance not only does the lid lag but it actually retracts, an effect due to contraction of the levator muscle, just as the internal rotation of the globe is due to contraction of the internal rectus muscle. These contractions and constriction of the pupil are the result of indiscriminate regeneration of oculomotor nerve fibers. During the process of healing, many of the regenerated axis-cylinders enter the empty nerve sheaths, passing to muscles other than those which they originally supplied. The net result is that nerve impulses meant predominantly for one muscle are shunted to all muscles innervated by the regenerated nerve, so that all the muscles contract simultaneously. Sometimes the axis-cylinders of one branch regenerate only in paths to one or two other muscles, so that the contractions en masse are not seen.

The problem of nerve regeneration and recovery of function of transected oculomotor nerves has been investigated by one of us (M. B. B.) in collaboration with Dr. Fulton at Yale University. Complete results will be published at a later date. In brief, it was found that in every monkey or chimpanzee in which the oculomotor nerve was cut

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1. Bender, M. B.: The Nerve Supply to the Orbicularis Muscle and the Physiology of Movements of the Upper Eyelid, Arch. Ophth. **15**:21 (Jan.) 1936. Bielschowsky, A., in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1907, vol. 8, pt. 2, chap. 11, p. 202; Lectures on Motor Anomalies of the Eye: II. Paralysis of Individual Eye Muscles, Arch. Ophth. **13**:33 (Jan.) 1935.

regeneration had occurred after a period of several months. Retraction of the upper eyelid and inward rotation of the globe were present whenever the normal eye looked downward, outward or, sometimes, upward. Vertical movements of the globe as part of mass contraction of all the ocular muscles were not seen, because the inferior oblique, the inferior rectus and the superior oblique muscle are antagonistic. When these muscles contract simultaneously, no gross movements result. Thus far, abnormally associated movements of the paralytic sphincter pupillae have not been observed in the experimental animal.

Section of the regenerated oculomotor nerve abolished all the abnormal movements in these animals, and the ophthalmoplegia reappeared. Analogous to observations made on regenerated oculomotor nerves were those made after experimental section of facial nerves. The results were almost identical. All experimental data seem to support the theory that the abnormal mass movements which occur after section of nerves are the result of faulty peripheral nerve regeneration and are not due to faulty nuclear regeneration as suggested by Fuchs.[2]

SUMMARY

A case is described in which a patient who had recovered from oculomotor ophthalmoplegia showed abnormal movements in the upper eyelid, the internal rectus muscle and the pupil. All three abnormal movements occurred simultaneously and in association with downward gaze. These movements are similar to those found after experimental section of oculomotor nerves in monkeys and chimpanzees. The abnormal movements are due to faulty peripheral nerve regeneration.

. 2. Fuchs, E.: Association von Lidbewegung wit seitlichen Bewegungen des Auges, Beitr. z. Augenh., 1893, no. 11, p. 12.

SOME NEW CONCEPTIONS REGARDING EGO-CENTRIC VISUAL LOCALIZATION

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The fact that one always sees things definitely placed in space in relation to oneself is so obvious that one rarely considers the mechanism of the appreciation of the spatial positions. More rarely does one consider the fact that such appreciation of position is a purely subjective phenomenon. This is occasionally impressed on one by discrepancies between the position of an object as determined by the eyes and that determined by tactile-proprioceptive sensation or objectively determined position. This discrepancy appears in cases of recent or fresh paralysis of the extra-ocular muscles which causes binocular diplopia. Immediately after the paralysis there is confusion because of the discrepancy, followed by gradual readjustment as the subject learns to disregard the falsely localized image. The objective interpretation of the relative positions of the double images in such cases, based on the simple laws of monocular projection, is well known, but analysis of the subjective phenomena involved has been neglected, primarily because of an inadequate understanding of the subjective aspects of localization under normal conditions of ocular function. Similarly, there has been a lack of understanding of the subjective phenomena of so-called anomalous correspondence of squints which has led to many erroneous conceptions in an attempt to explain the anomaly. It is the purpose of this paper to lay the foundation for a more correct interpretation of these phenomena by an analysis of subjective egocentric localization under normal conditions. An analysis and interpretation of the sensory anomaly in squints will be presented in a later publication.

Subjective egocentric, or absolute, localization (von Graefe's "subjective orientation") is perception of the positions of seen objects in relation to the observer's body image. The term may be used also to denote the visually perceived positions of objects in relation to the body image. The body image is the instantaneous subjective spatial representation of the observer's own body given as a composite of the representations by the various sensory systems, most of the parts visible to the observer himself being represented visually while the nonvisible parts are represented by tactile, proprioceptive and gravistatic sensa-

tions. The correct fitting together of these representations depends on subjective correlation of the various sensory systems.

A characteristic of vision is that a stimulus affecting the retina is not perceived as stimulation of a definite part of the body but is perceptually externalized and is identified directly with its apparent source. This externalized representation of the stimulus is localized subjectively with respect to the body image, especially the head portion, without particular reference to, or consciousness of, the exact anatomic site of the stimulation. The retina, therefore, cannot be said to have a local sign as a part of the head image but has instead a subjectively externalized intrinsic local sign which is spatially correlated with the head image.

What little evidence there is points to an innate origin of this spatial correlation between the externalized visual representations and the head image rather than its acquisition through individual experience. Young infants, before they are able to grasp seen objects, turn the head or eyes toward a source of light once the latter has been "found" by the fovea, which must indicate that the source is localized subjectively, at least in a crude fashion, by foveal fixation. Foveal fixation and thus probably foveal localization are present before localization develops in the periphery of the visual fields. The so-called developmental unfolding of the visual field postnatally, as evidenced by the infant turning its eyes or its head toward a peripheral source of light, is probably the development of subjective localization of the periphery rather than the actual beginning of the perception of stimuli by the peripheral visual elements. The fact that simple awareness of stimuli in the peripheral field appears to be a more highly developed function than accuracy of localization suggests that the former is present fundamentally. Of course, in the infant there is no definite indication of simple awareness of particular stimuli except in behavior dependent on localization.

In normal binocular vision the two eyes seem to function subjectively as a single organ. That is, one is not aware of a visual stimulus affecting two separate organs, since only a single source is seen. Binocular vision is generally conceded to be an innate faculty. The singleness of binocular vision is singleness of externalized representation. Verhoeff [1] gave the name "unification" to this and explained it by his theory of replacement, according to which the unified binocular percept is a composite, resembling a mosaic, of the respective monocularly seen elements. Thus the percepts arising from corresponding points are not seen simultaneously, but one replaces the other. Which of the two is consciously seen is determined by their relative attention

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1. Verhoeff, F. H.: A New Theory of Binocular Vision, Arch. Ophth. **13:** 151-175 (Feb.) 1935.

values; the one which arouses attention of the higher degree is given precedence. A fundamental for such a theory is that percepts arising from corresponding points always are localized in the same spatial position with respect to the head image; otherwise replacement would result in incongruities. In fact, this is the subjective basis for the conception of correspondence. Thus, the representations of corresponding points are interchangeable without subjective alteration of the spatial nature of the binocularly unified image.

In agreement with the theory of replacement, therefore, normal binocular visual localization may be conceived to be the result of the proper fitting of the separate, monocularly originating elements into a common spatial scheme or framework, the localization of which is innately correlated with the body image, especially the head image, so as to agree with that given by the other senses. The spatial representations of corresponding retinal points may be said to stand in a mutual relationship to a single subjective localization. This will be considered more fully later.

Subjects with normal binocular vision and without deviation of the visual axis of either eye under cover will see objects of foveal fixation in the same position monocularly, when one eye is covered but not closed, as binocularly. If, however, there is lateral deviation of the axis of the eye which is covered, the localization of objects seen monocularly will appear displaced, relative to their binocular localization, in the direction of deviation of the axis of the covered eye. A similar phenomenon is associated with change of fixation from near to distant objects, and vice versa. When, for instance, one eye is covered but not closed and the other eye is fixed first on a distant object, and then changes fixation to a near object which is alined monocularly with the distant one, the whole monocularly seen field will appear to shift its position toward the side of the uncovered eye. This depends on the change in convergence of the visual axes which is associated with change of fixation from distant to near objects. If instead of simply being covered one eye is closed voluntarily with the lids, these shifts in localization of the monocularly seen field either do not occur or are of slight, variable degree. One intentionally can vary these phenomena by controlling the position of the nonobserving eye whether it is covered or closed, particularly in the former case if there is enough phoria in the proper direction to maintain the position of the covered eye.

In these phenomena the subjective localization of the seen field is obviously influenced by the actual direction of the visual axis of the covered eye. This leads to the conclusion that whether an actual observation is made monocularly or binocularly, as long as both eyes are

open the individual positions of the two foveal visual axes are simultaneously and jointly associated with a single definite subjective localization.

Association of stimulation of corresponding points with a single subjective localization, therefore, makes it unnecessary to distinguish consciously between the respective monocularly seen parts of a binocularly unified image, and also, by virtue of replacement, makes it impossible to tell under ordinary circumstances whether an object is imaged on only one retinal point or simultaneously on both of a pair of corresponding retinal points. The fixation intent is normally directed toward binocular fixation and is therefore subjectively associated with simultaneous foveal fixation in both eyes. Consequently, regardless of whether an object is imaged on one or on both foveae, it is localized subjectively as if imaged on both, and the localization is that which is associated with the instantaneous positions of both foveal visual axes.

The phenomena associated with paralysis of the extra-ocular muscles and with passive rotation of the eyeball indicate that the actual position of each foveal visual axis is correlated with a definite innervational status of the extra-ocular muscles which effect the rotational position of the eye. For example, after paralysis of the lateral rectus muscle, attempts to rotate the affected eye outward are at first attended by a subjective shift of the whole field seen by this eye in the direction of, and by an amount approximately equal to, that of the intended transfer of foveal fixation. Thus the objects imaged by the fovea are localized subjectively in the position of the intended foveal fixation as represented by the muscular innervation which would normally accomplish it.

In the case of passive rotation of the eyeball, by external means or as occurs reflexly in Bell's phenomenon, the objects imaged by the fovea, and the rest of the field, according to its local sign arrangement about the former, are seen in the localization given by the intended innervation of the extra-ocular musculature. Since this is unchanged during passive rotation, the succession of objects imaged by the fovea is seen as a succession of objects moving through the given localization in a direction opposite to that of the displacement of the foveal visual axis. Or, since the field is seen as a whole, it appears to move as a whole in this direction.

One is conscious not of the actual positions of the foveal visual axes as such but only of the single subjective localization with which they are jointly associated. Their individual positions both enter into the determination of this localization, and for this purpose it is apparently necessary for a conscious image to be obtained from only one fovea. The innervational status of each extra-ocular musculature, then, must be the means of subjectively, but not consciously, representing, in

physiologic terms, the position of its foveal visual axis. According to this, regardless of the actual position of the foveal visual axis, the position which enters into the subjective localization is that which the innervational status of the extra-ocular musculature represents as present.

The localization with which we are concerned is essentially a gross one with relation to the body image. It involves in a similarly gross fashion depth perception, that is, the distance of seen objects from the body image, especially the distance of the object of fixation. It thus utilizes primarily the same factors as monocular depth perception, since only the localization with which corresponding points are correlated is involved, and binocular parallax therefore does not serve it directly. The latter, however, may furnish clues to the size-to-distance relationship and thus indirectly aid in the subjective localization.

Certain phenomena indicate that subjective representation of the relative positions of the foveal visual axes is a factor in subjective egocentric distance localization. The phenomena mentioned consist of changes in the apparent size of objects on inducing so-called passive convergence or divergence as by slipping prisms before the eyes. Passive convergence results in an apparent diminution of size and consequently a judgment of remoteness. Passive divergence causes the reverse phenomenon. As was pointed out by Verhoeff,[2] the only logical interpretation of these phenomena is that the subjective representations of the positions of the visual axes are associated directly with the idea of size and through this with the idea of distance. Since the visual angle subtended by the object actually remains constant, the apparent size of the object is that which, for this visual angle, is correlated with the instantaneous innervational statuses of the extra-ocular musculatures representing the positions of the foveal visual axes.

On considering these phenomena, it is apparent that one does not see distance or intervening space as such but sees only the apparent sources of one's retinal images. One's impression of visual space is thus directly dependent on, and is in terms of, one's perceptions of the spatial nature of its contents, which invariably exist for one in three dimensions. This means not that one always localizes the objects seen correctly, that is, in agreement with objective localization in three dimensions, but that one's idea of the spatial existence of seen objects is always three-dimensional, whether or not one can express it accurately in objective terms. Seeing something always implies seeing it somewhere in a three-dimensional spatial relationship to oneself, whether it is observed with one eye or with both eyes.

2. Verhoeff, F. H.: A Theory of Binocular Perspective, Am. J. Physiol. Optics **6**:416-448, 1925.

In normal binocular visual localization, then, the following fundamental correlations are present: (1) the correlation between the fixation intent and simultaneous binocular foveal fixation; (2) the correlation between the position of each foveal (fixation) visual axis and the innervational status of the respective extra-ocular musculature, this position being represented subjectively in terms of the innervational status; (3) the simultaneous, joint correlation between the positions of the two foveal visual axes, as given in these terms, and a single subjective localization in three dimensions, the third dimension, or depth, involving (4) a further correlation between these subjective innervational representations and the size, and thus the distance, of seen objects. By virtue of the consistent orientation of other points in each field about the fixation point in accordance with their local signs, these correlations may be extended to corresponding points in general. I subscribe to the theory of an innate origin of these fundamental correlations on the basis of phylogenetically acquired cerebral nervous connections. The rationale for this belief lies in the analogy to other phylogenetic changes in the central nervous system which serve binocular vision. These are the connections of the motor nerves serving conjugate and disjunctive movements and the partial decussation of the optic nerve fibers which serves the overlapping of homonymous halves of the visual fields.

RETINAL CORRESPONDENCE

One may now examine more critically the relations generally existing between so-called corresponding points. As was emphasized previously, normal correspondence between retinal points in the two eyes requires that percepts, arising from these points, always be localized interchangeably in the same spatial position in relation to the head· image. Correspondence thus means a fixed mutual association with a single subjective localization in three dimensions. Its usual definition in terms of subjective direction, whether referred to the so-called cyclopean center of directions or any other point in the head image, is inadequate, since the term direction involves only two dimensions. Moreover, to say that normally corresponding points are those points represented subjectively by identical directions is incorrect, for, in the first place, a single retinal point is represented subjectively not by a direction but by a spatial position in three dimensions as long as one has cognition of three-dimensional space, and, in the second place, any retinal point in one eye, when the other eye is not closed consciously, is not normally correlated independently with an individual subjective localization, even though the other eye is completely excluded from the observation. As was stated previously, there is normally a simultaneous, joint correlation between the subjective representations

of the positions of the foveal (fixation) visual axes and a single subjective localization in three dimensions. Likewise, the representations of the visual axes of any other pair of corresponding points are, through their local sign relations to their respective fixation axes, similarly correlated with a single subjective localization. These representations of the visual axes, however, cannot be said to indicate independently individual subjective localizations. They are unconscious representations in physiologic terms and are correlated not independently but jointly with one subjective localization. Consequently, in normal binocular localization there is no independent subjective localization by either eye alone while the other eye is not consciously closed, even though the latter is completely excluded from observing.

OCULAR DOMINANCE AND THE CYCLOPEAN EYE

The so-called cyclopean eye, or the center of visual directions supposedly lying about midway between the two eyes and a little behind them, is purely a hypothetic schema based on the fact that in persons with normal binocular vision both monocularly and binocularly alined objects appear to lie on a line which passes about through this position in the head image. The objects may be alined with one eye alone, the other being open but excluded by a shield, or one may be alined in the fixation axis of one eye and the other in the fixation axis of the other eye, so that each object is seen by only one eye. Binocular alinement is achieved by changing fixation from one object to the other. The understanding of the phenomena of monocular alinement is best approached through analysis first of binocular alinement.

Any one with normal binocular vision can demonstrate to himself the fact that binocular alinement of objects apparently represents a fixed subjective state in the conjugate displacement of the eyes for various degrees of convergence. Thus, when one looks binocularly from a distant to a near object, the two objects will appear to be alined with the observer when there is no subjective change in the conjugate displacement of the eyes on changing the fixation. This phenomenon I shall call the binocular alinement test. From what has been said previously, disjunctive and conjugate movements of the eyes are not consciously represented as such but are represented only in terms of changes in the three-dimensional spatial values of seen objects in relation to the body image, so that absence of subjective change in the conjugate position of the eyes results in changes in convergence being represented consciously as changes in size and distance or depth in a line with the "center" of the observer.

This subjective center is a part of the body image as a whole. As a result of the bilaterally symmetrical arrangements of the parts of

the body, there is a tendency, in the absence of a specifically different intent, to refer subjective, egocentric localization to about the median longitudinal axis of the body image. This axis of reference may be spoken of as the axis of balance for the symmetrically arranged parts, since, at any level, it represents approximately the position of the center of balance. As a whole, it represents also the subjective axis of inertial balance for torsions of the body or its parts. It forms the basis of the subjective median longitudinal plane and thus the origin for the subjective coordinate system of the body image. Objectively, it obviously is not a single straight line but will have various shapes depending on the characteristics of the body.

It might be supposed, with maintenance of the same degree of innervation for conjugate deviations subjectively, that disjunctive movements of the eyes would be objectively symmetrical, that is, the angular rotations of the eyes would be equal but opposite. This supposition, however, ignores certain phenomena on which I believe so-called ocular dominance is based. The usual tests for dominance involve the selection of one eye or the other for monocular alinement or sighting. The explanation for the dominance and its relation to cortical dominance are not obvious from these tests, since one part of each eye is represented sensorially in one half of the brain and the other part of the eye in the other part of the brain and each eye is also subject to motor control from both halves of the brain.

In the binocular alinement test it is common for the objects which are subjectively alined with the axis of balance of the observer to be alined objectively with some point nearer to one eye than to the other. A similar phenomenon has been observed in the alinement of the subject's concealed finger, as when it is under a table or a board, with an object seen binocularly in the distance. The latter test has been used extensively to determine the objective position of the subjective median vertical or longitudinal plane. What the subject actually does is simply to compare the visual localization with the tactile-proprioceptive localization of this plane, for both are subjective. The visual localization coincides with the subjective visual, straight-ahead position which represents physiologically a null point in the innervation for voluntary conjugate movement of the eyes to either side. Its objective position would then depend on factors not under voluntary control and thus chiefly anatomic and physiologic.

Conjugate deviations of the eyes to one side or the other are initiated voluntarily in the portion of the cerebral cortex contralateral to the side of the deviations. It is reasonable, then, to assume a one-sided motor dominance of ocular movements which is analogous to the one-sided dominant development of voluntary motor functions of the rest

of the body contralateral to the dominant portion of the cortex. With dominance of the left side of the cerebral cortex, then, an unconscious hyperactivity of the right conjugate deviators of the eyes should be expected.

If anatomic anomalies of the ocular musculature or orbits are disregarded, the actual position of the subjective visual, straight-ahead position in general would depend on the balance of muscular tonus between the right and left conjugate deviators at the null point of conscious innervation. Hyperfunction of the right deviators would then result in the null point coinciding with more or less deviation to the right and consequently giving a subjective visual, median, longitudinal plane objectively to the right of the actual median plane. Conversely, with right cortical dominance and sinistrality, the left conjugate deviators would be dominant and the subjective visual median longitudinal plane would be objectively to the left of the actual median plane.

The binocular alinement test in conjugate positions other than the straight-ahead position reveals the same effect of motor dominance. Thus, for right or left conjugate positions, binocularly alined objects will be found objectively to be alined more toward the dominant motor side relative to the alinement given by objectively symmetrical convergence. The latter alone would give an alinement toward the side of the actual deviation, as may be shown geometrically. The effect of the dominance, then, is to cause the alinement to be objectively toward the dominant motor side of the alinement given by objectively symmetrical convergence. Any one can test this on himself by noting the positions of the physiologically doubled images of one of the two alined objects relative to the other binocularly fixed object. The alinement by symmetrical convergence would be represented by the binocularly fixed object appearing approximately midway between the two images of the other one, whereas a deviated alinement resulting from unilateral motor dominance would be represented by the former object appearing nearer to that one of the doubled images which belongs to the eye on the side of the motor dominance.

The physiologic effect of the conjugate motor dominance on convergence in the absence of conscious change in the conjugate innervation is thus an objective asymmetry toward the side of the dominance.

My interpretation of so-called ocular dominance according to conjugate motor dominance is that, generally, the directing or dominant eye is the one for which the alinement seems to agree best with the subjective binocular alinement determined by the given conjugate position of the eyes. In this connection it must be borne in mind, from what was said earlier, that even though only one eye is doing the observing while the other is open but excluded, the subjective repre-

sentation of their conjugate position and thus the conscious localization of the seen objects are determined by the correlation between the representations of their respective muscular innervations and a given localization.

A test which I devised several years ago demonstrates, in a manner different from the sighting test, to which side of the head image visual egocentric localization is referred. It depends on the well known fact that, under controlled conditions of observation, such as dark surroundings, a subject with normal, binocular vision who has both eyes open but one excluded by an opaque screen placed in front of it cannot tell which of his eyes is doing the observing. Under these conditions, then, one eye is shielded, and a slender rod is aimed by the examiner either at the inner or the outer canthus of the observing eye. The subject is directed to indicate with his finger at what part of his head the rod appears to point. If it appears to point toward the nonobserving eye, when it actually is aimed at the inner canthus of the observing eye, or if it appears to point to the outer side of the observing eye when aimed at its outer canthus, reference of visually perceived position is assumed to be predominantly on the side of the observing eye, and that eye is termed the "reference eye." If, when the rod is aimed at the inner canthus of the observing eye and the subject indicates that it is pointing toward the outer side of the nonobserving eye, or, when aimed at the outer canthus of the observing eye, it seems to point between the eyes, the nonobserving eye is the reference eye. The results of the test on a number of subjects indicated with few exceptions that the same eye was used for sighting as for subjective reference. Even when the eye used for sighting was not on the side of the motor dominance, the reference eye usually agreed with the sighting eye rather than with the side of motor dominance. These results indicate that the factors determining the sighting eye probably operate also to determine the reference eye.

The customary use of one eye for monocular alinement does not lead directly to the conscious selection of it under the conditions of the sighting or the reference tests but only to the consistent reference of egocentric visual localization to its side of the head image. The basis, then, for the selection of this eye by the sighting test is the better agreement of the apparent subjective visual localizations through it with the localizations determined, respectively, by the conjugate position of the eyes and by tactile-proprioceptive sensations. Thus, in the sighting test as commonly performed, a disk with a hole in it, or a tube, is held by both hands, and a distant object seen binocularly is "sighted" monocularly through it. The position of the distant object is thus given by the conjugate position-localization of the eyes, and the

position of the disk or of the tube is given by the manual tactile-proprioceptive mechanism. The hole or the tube is placed before the eye, which gives thus an apparent egocentric alinement in closest agreement with that given by the aforementioned localizations. If the nonsighting eye is inadvertently used for such tests there is a definite sense of confusion or disagreement in the localizations, and either the manually determined position of the disk or the tube seems wrong or the subject immediately realizes that a particular eye is being used.

Other factors also enter into the choice of the sighting eye, especially the relative visual acuity of the eyes and habit developed in connection with certain tasks. Certain muscular dysfunctions may also play a part. The apparent so-called dominance, determined by the sighting test, is thus simply the result of a selection guided by either a concomitance of these various factors, including the actual conjugate motor dominance, or a predominance of one or more of them.

Of course, mixed types of dominance also would be expected to occur, probably on the basis of a mixed cortical dominance. There is no reason why every person should have complete right or left cortical dominance for all motor activities. Initial training and necessity frequently cause mixed dominance such as is present in the case of left-handed persons who learn to play golf with right-handed clubs or cases of right-handed persons who have a dominance of the left eye in other ways but who aim a rifle by sighting with the right eye.

The phenomena of monocular alinement are based on the same fundamental principles as those of binocular alinement. The objects, which are observed monocularly, whether one is in the fixation axis of one eye and the other in that of the other eye or both are in the same fixation axis, have their egocentric subjective localizations given according to the four correlations previously described. In monocular alinement both conjugate and disjunctive innervations are held in their existing subjective state. The only clue, then, to the relative egocentric localizations of the objects is in their size-to-distance relationship, the nearer one appearing the larger and being correctly interpreted as nearer, or if they are both on the same fixation axis the additional factor of the nearer one obscuring all or part of the distant one confirms this judgment. As was brought out in discussing binocular alinement, changes in convergence in the absence of subjective change in the conjugate position of the eyes are represented as changes in size and depth distance in a line with the subjective center, or axis of balance, of the observer, objective deviations occurring toward the side of the conjugate motor dominance irrespective of asymmetry of convergence. Thus, this line represents the locus of subjective egocentric localizations with which various convergent positions of the eyes are correlated when there

is no subjective change in their conjugate position. Consequently, in monocular alinement, the subjective egocentric (depth) localizations determined by the size-to-distance relationship are correlated with their respective convergence innervations and seen in the locus of positions determined by these, thus apparently in a line with the subjective axis of balance of the observer.

According to these interpretations, it is difficult to see any basis for assuming a primary center of visual directions as implied by Hering's rule. If one were to limit oneself to the use of directions alone without three-dimensional localization in interpreting the phenomena of alinement, one would have to assume a primary center of reference for these directions. Thus, if one accepts identity of directions as the criterion for retinal correspondence, then, as Helmholtz [3] has stated, one must assign a center of reference for the directions. But if one considers the fact that the monocularly observed objects are individually localized subjectively in three dimensions, the position in the head image of this so-called cyclopean center of directions, as indicated by the phenomena of alinement, is seen to be simply the physiologic consequence of the fundamental subjective correlations which determine subjective egocentric localization. It is thus not a fundamental subjective center of reference for visual direction but is merely the result of the visual interpretation of the subjective axis of balance of the body image.

SUMMARY

Subjective egocentric visual localization is the determination of the position of seen objects in relation to the body image of the observer. It is accomplished by means of four fundamental correlations of innate origin: (1) the correlation of the fixation intent with simultaneous, binocular foveal fixation; (2) the correlation of the positions of the individual foveal (fixation) visual axes with the innervational states of the respective extra-ocular musculatures, these positions being represented subjectively in terms of the innervational states; (3) the simultaneous, joint correlation of the positions of the individual foveal (fixation) visual axes, as given in these terms, with a single subjective egocentric localization in three dimensions, and (4) the correlation between these innervational representations and the apparent size of objects, which indirectly furnishes the third dimension, or depth, through size-to-distance relationships.

Subjective egocentric visual localization is always three-dimensional, whether the observation is made monocularly or binocularly and whether

3. Helmholtz, H. L. F.: Helmholtz's Treatise on Physiological Optics, translated from the German edition and edited by James P. C. Southall, Rochester, N. Y., The Optical Society of America, 1925, vol. 3, p. 243.

or not there may be agreement with objective localization or accuracy of expression of the localization in objective terms.

Retinal correspondence denotes a functional relationship between individual retinal points in the two eyes such that percepts originating from them have an innately fixed mutual correlation with a single subjective egocentric localization in three dimensions. Definition of correspondence in terms of direction is incorrect as well as inadequate.

The phenomena of binocular and monocular egocentric alinement are based on the four fundamental correlations involved in subjective egocentric localization with the additional factor of true unilateral motor dominance. The latter exerts its effect through hyperactivity of conjugate deviation toward the side of the motor dominance, therefore contralaterally to the dominant half of the cerebrum. The alinement is the result of the maintenance of conjugate deviation in the existing subjective state and is determined by the locus of subjective egocentric localizations which are correlated through the size-to-distance relationship with the various convergence innervational states for this subjective state of conjugate deviation. Because the localizations are all in relation to the body image and because of the approximate symmetry of the parts of the latter about the subjective axis of balance to which localizations in general are referred, this locus is directed toward the axis of balance also. The point toward which it actually appears to be directed represents, therefore, not a primary center of visual directions (the so-called cyclopean eye) but merely the interpretation of the subjective axis of balance in terms of subjective egocentric visual localization. Because of the effect of unilateral motor dominance on conjugate deviation, this point is displaced toward the side of the motor dominance.

A new interpretation is given so-called ocular dominance, based on the effect of unilateral motor dominance on conjugate motor deviation. The dominant eye as selected by the sighting test is the one for which the alinement thus determined usually agrees best with the subjective alinement based on the given conjugate position of the eyes. Thus, if the conjugate position of the eyes has suffered a deviation to one side as the result of motor dominance on that side, sighting with the homolateral eye will give an alinement which agrees better with the subjective egocentric positions of the target and the sight than that given by sighting with the other eye. Habit or training and the relative visual capacities of the two eyes are also factors in determining the sighting eye.

Clinical Notes

A CASE OF LYMPHOCYTIC MENINGITIS (?) WITH OCULAR FINDINGS

GERALD D. SPERO, M.D., DETROIT

Benign lymphocytic meningitis seems to be on the increase. The disease is peculiar in that it is characterized by a severity of symptoms that is surprising, in view of the fact that recovery is prompt and complete. The condition occurs chiefly in younger persons.

REPORT OF CASE

History.—S. A., an Italian youth aged 15, was referred to me on Aug. 18, 1936. His chief complaint was photophobia. With the exception of scarlet fever a number of years previously, he had always enjoyed good health. About one week prior to my seeing him he began to complain of extreme photophobia. This was accompanied by headaches, with no particular localization. These symptoms were constant. Dark glasses did not help. Salicylates helped only a little.

Examination.—At this time a general examination showed nothing of importance. The ocular examination revealed the following:

Vision in each eye was normal. Refraction was carried out with the employment of homatropine, and with the eyes under the influence of drops the patient accepted a + 0.75 D. sph. combined with a + 0.25 D. cyl., ax. 90 for each eye. With this correction, vision in each eye became 20/20 + 4. The extra-ocular movements were normal. The pupillary reflexes, media and tension were normal. Tests of the ocular muscles showed nothing of importance. The right disk was slightly hazy nasally, but no swelling was present. However, the entire left disk was swollen, to the extent of from 0.5 to 1 D. The swelling was confined entirely to the disk. The veins around the disk were somewhat engorged, the arteries being normal. The picture was suggestive of early choked disk. The field for color and that for form were normal in each eye. No central scotoma was present in either eye. The record of the ocular findings was turned over to the attending physician, and he in turn called in a neurosurgeon, Dr. S., for consultation. The latter advised hospitalization.

Subsequent Findings and Course.—The subsequent findings and the course in the hospital were as follows: Shortly after admission the temperature rose to 102 F., and the lymphatics (axillary, inguinal and cervical) became slightly swollen. During the first few days the patient vomited several times (the vomiting being projectile in type). In addition to the photophobia and headaches, dizziness was noted. The patient also complained of mild pains in the muscles of the calf. The pulse rate on admission was 66. The respiratory rate was 18.

Shortly after admission a lumbar puncture was done, and the fluid was found to be under some pressure and clear. No organisms could be demonstrated. Nine hundred cells were present. Of these, 72 per cent were lymphocytes and 28 per cent polymorphonuclears. The colloidal gold chloride test showed nothing of importance. The Kahn reaction was slightly positive. The test for chlorides

showed 726 mg. of sodium chloride per hundred cubic centimeters. The globulin content was slightly increased. The sugar content was found to be 0.073 per cent.

Examination of the blood on admission showed the hemoglobin to be 95 per cent and the red cell count to be 4,720,000. The white cell count was 14,900. The differential count was as follows: stab cells, 8 per cent; segmented cells, 70 per cent; lymphocytes, 20 per cent; large monocytes, 2 per cent. The urine had a specific gravity varying between 1.012 and 1.008 and contained no albumin or sugar. On admission a trace of acetone was found, but subsequently acetone was absent.

On October 23 the pressure of the spinal fluid was still somewhat elevated, but the number of cells had dropped to 130, and of these 92 per cent were lymphocytes and 8 per cent polymorphonuclears. The complaints had started to lessen in severity. On October 24 the spinal fluid was clear and under no pressure. On October 26 the patient was free from all complaints, and since the spinal fluid was normal he was sent home. Several weeks after his discharge from the hospital the patient felt fine, had gained some weight and returned to work. It seems that his complaints started to disappear with the first lumbar puncture and were gone the day after the third one. When he was last seen (several weeks after discharge from the hospital) the disks had become completely normal.

COMMENT

Differential Diagnosis.—Encephalitis: The presence of choked disk and the absence of elevation of the level of sugar in the cerebrospinal fluid speak against encephalitis in the case reported. In some cases, though, differential diagnosis between encephalitis and lymphocytic meningitis may be difficult.

Poliomyelitis: This condition may be present, and probably frequently is, without paralysis, causing signs of general infection with or without localizing signs in the central nervous system. In the case reported here, however, the cell count of the spinal fluid at the original examination was much higher than that found in infantile paralysis.

Tuberculous Meningitis: Tuberculous meningitis is easily ruled out in the case reported in this paper by the fact that the patient recovered, and it was not seriously considered in the acute stage because of the normal sugar content of the spinal fluid, the absence of the tubercle bacillus in the spinal fluid and lack of evidence of any systemic tuberculosis.

Other Diseases.—In epidemic meningitis, as well as in all bacterial forms of meningitis, examination shows the causative organisms in great profusion and without difficulty. Further, these conditions are usually characterized by higher cell counts and the presence of a much larger proportion of polymorphonuclear leukocytes than was found in the case just reported.

Syphilis of the central nervous system presents a manifold and often bizarre clinical picture, depending on the extent of involvement and the areas affected. In syphilis sufficiently active to produce the symptoms noted in the case reported one would certainly expect a strongly positive serologic reaction of the spinal fluid and a

more atypical colloidal gold curve than was obtained, even though the serologic reaction of the blood might be negative. Further, the age of the patient and his antecedent history speak strongly against this diagnosis.

A clinical syndrome known as meningitis aseptica, which is an inflammatory process of the meninges due to nonbacterial causes, has been described in the literature. It has followed injections of certain irritating medications and serums, as well as saline irrigations of the spinal canal. None of these factors, however, was responsible in the present case.

CONCLUSIONS

For want of a better term I am calling the condition in the case reported in this paper acute lymphocytic meningitis. Others call it aseptic meningitis. In any event, the disease is mild, and a good prognosis can be given. Many of the patients show no ocular findings. In my case a choked disk was present. Other authors have reported a paretic squint, which usually clears up. Clinically, the outstanding complaint is photophobia. Headaches and dizziness may also be present. Early in the disease an excessive number of polymorphonuclears may be present, and lymphocytosis may exist later. The total protein content of the spinal fluid is increased, and globulin is invariably present. There is only a slight reduction in the amount of dextrose and chlorides in the spinal fluid—an important feature distinguishing this condition from tuberculous meningitis, in which a definite and marked diminution of the amount of chlorides and dextrose is always present. The average duration of the disease is seven days. The temperature falls by lysis, and, as a rule, complete recovery takes place in two or three weeks. This condition is now generally regarded to be due to a distinct virus recently recovered (in 1934), and thus the term aseptic meningitis would, scientifically, be a misnomer. Examination of the spinal fluid is necessary to establish a diagnosis. The treatment is largely symptomatic. Repeated spinal punctures relieve the photophobia and headaches. The disease affects mostly young persons (those from 20 to 28 years of age), and, as a rule, there is no history of an infection of the ear or the nasal sinuses. This clinical syndrome should be borne in mind. As I look over past histories it seems to me that some of the conditions previously reported might have belonged to this group.

A NEW CALIPER
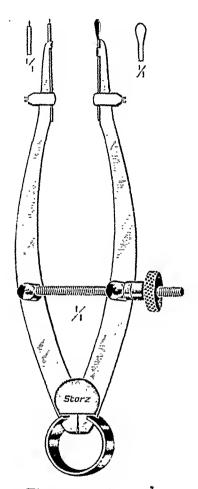
John Green, M.D., St. Louis

Accurate measurement from the line of insertion of any rectus muscle to the point on the sclera where it is desired to recede or advance that muscle is necessary. For this purpose a number of calipers have been designed. I have not been satisfied with any so far placed on

the market. One type, designed on the principle of a slide rule, measures accurately the desired distance, but the projecting bars are dull and do not give an opportunity to indicate by a delicate scratch mark the position on the sclera for the insertion of the sutures. Another type is a small caliper with dull points neither of which serves the particular purpose desired.

I have had an instrument maker construct a caliper one shank of which carries a thin shaft of steel which terminates in a very fine sharp point. This point cannot penetrate the sclera, as it is guarded by the

The new caliper.

shaft. The other arm of the caliper carries a small round-ended knife, which can be removed for sharpening, if necessary.

In use the point and the knife are separated to the desired distance (e. g., 5 mm.) by means of the adjusting screw.

The sharp point penetrates the upper layers of the sclera at either the upper or the lower border of the tendon at its insertion, and a tiny scratch mark is made on the sclera with the round-ended knife. This scratch mark is fine but sufficiently distinct so that when the sclera is freed of blood it is possible to place a suture precisely.

The caliper has been found of great use in Curdy's recession operation and also in Curdy's advancement operation.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

SOME PROBLEMS AND PROCEDURES IN REFRACTION

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ROCHESTER, MINN.

Refraction calls for a technic of painstaking mathematical accuracy combined with clinical judgment, that valuable art the anlage of which is a physiologic point of view—the ability to visualize the human machine as a whole, each part in relation to the other, and to see the mechanism at work, sensing dysfunction, incoordination, conflicts, defects and organic troubles in their true relation and importance to the patient as a living mechanism. In other words, the ophthalmologist should not confine his interest, knowledge and attention to the narrow limits of his special field so that he goes off on a tangent and fails to comprehend the theme of the complete clinical picture. Diagnosis and treatment in a specialty must fit in accurately with the numerous and varied factors making up the many jagged parts of the complete clinical picture. Failure to take this into account is perhaps the outstanding criticism of specialization. After all, the ophthalmologist is the physician practicing ophthalmology.

With the foregoing considerations in mind, it may be stated that refraction has for its major purpose the alleviation of symptoms of ocular distress by the proper fitting of glasses. Local medicinal treatment, orthoptic exercises and surgical measures are necessary and are valuable adjuncts when intelligently used. In the prescribing of glasses, science and the art of practicality must be deftly mixed, it being ever remembered that the acid test is the patient's approval. The prescribed glasses must give clear, ready vision and enable the patient to carry on his work and play with comfort and with a minimum of fatigue. To accomplish this constitutes the fine art of refraction.

In the practice of refraction much trouble and dissatisfaction can be avoided by keeping in mind certain pertinent relationships existing between the ophthalmologic, general medical and neurologic aspects of the case. The ocular symptoms of which a patient complains may

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be but reflex or mirrored disturbances arising from a disordered mental or physical state. The eyes, by virtue of their origin from, and their intimate connections with, the brain and the central nervous system, are prone to reflect disease and functional disorders of these structures. Neurasthenia, psychoneurosis, traumatic neurosis, hysteria, mental inadequacy and similar disorders usually cause symptoms of ocular distress which glasses or treatment of the eyes will not cure. The prescribing of glasses for the unfortunate persons with such a disorder brings nothing but trouble for the physician. Increased intracranial pressure from tumors and abscesses of the brain causes decrease in vision, and glasses are not the answer; atrophy of the optic nerve and optic neuritis affect vision likewise, and the cause must be looked for elsewhere than in the eyes. The ophthalmoscope and the perimeter must be constantly employed to avoid the pitfall of attributing all disturbances of vision to an ocular cause. Toxic amblyopia resulting from tobacco, alcohol or some other poison or due to syphilis may be interfering with vision, and the ophthalmologist must be quick to note the presence of such a factor. Retinitis from arteriosclerosis, hypertension, pregnancy, anemia, diabetes, renal disease or some other condition may be causing visual difficulty. The asthenic, run-down and generally ill patient naturally has no ocular comfort. Those recovering from recent general surgical and medical treatment often complain of inability to use their eyes. All these and many other neurologic and medical disorders secondarily affect the eyes, and the alert ophthalmologist hesitates to prescribe glasses until such time as the patient has been restored to a reasonable degree of health.

Age and senility also bring about a decrease in ocular capacity. One should change glasses for aged persons with caution, as dissatisfaction with new glasses is the rule. It is not possible to enable these patients to use their eyes comfortably and as much as they wish with any glasses. Persons working at high tension, which creates nervous exhaustion, have much ocular fatigue, and glasses will not correct this. The local, social, economic and familial status must also be taken into consideration. Thus, when a patient comes in for refraction the ophthalmologist must consider all these various factors and attempt to evaluate the condition of the patient and consider whether it is feasible to undertake to give relief by ocular treatment or glasses. By such a differentiation much unpleasantness can be saved the ophthalmologist and serious trouble and dissatisfaction can be saved the patient.

A problem which has interested me for a long time is the basic difference clinically between various types of eyes. There is a sharp contrast in the visual capacity and reactions of the emmetropic, the myopic, the hyperopic and the astigmatic eye. One cannot expect the

same performance from all eyes. They differ from one another in their reactions and capacity, just as one person varies from another. It is important and useful to keep this in mind when evaluating ocular symptoms and attempting to prescribe for them. The clinically ideal eye is perhaps the eye which does not vary too far on either side of emmetropia and has not too much astigmatism—in other words, an eye near the normal in the curve for frequency distribution of refractive errors. Such an eye, whether it is slightly hyperopic or myopic, as a rule carries on its functions well.

The more eyes deviate, however, from the normal toward the higher degrees of ametropia and the more they are affected by marked astigmatism, the more difficult becomes their task of carrying on. Glasses, as is known, do not make eyes normal, nor do glasses enable eyes with marked errors of refraction to perform with the same ease as those eyes near normal. It is therefore natural to expect that markedly ametropic eyes, though well corrected, should give much more discomfort and have less capacity for ease of function. The normal eye is an effortless mechanism, and the possessor of such eyes is to be envied. The markedly ametropic eye, on the other hand, has to be whipped and spurred to make it accomplish its tasks. When, in addition, anisometropia and antimetropia exist, differences in the amount and the kind of error in the two eyes, the difficulty of proper ocular performance becomes even more exaggerated. In these markedly anisometropic and antimetropic eyes there exists an essential and intrinsic irritability which even the best of corrections usually fails to subdue completely. Persons with these eyes often get into a bad state ocularly and mentally through lack of understanding of the true nature of their ocular mechanism by themselves and by their ophthalmologist. Not obtaining relief, they relentlessly pursue comfort and go from physician to physician. Having tried numerous corrections and much varied advice and continuing to be uncomfortable, they naturally may assume that there is something radically wrong with their eyes. They come to feel that they are faced with eventual ocular disaster.

Although this seems a logical conclusion to them, one knows that it is not true. It is a false conclusion drawn through misinformation and lack of understanding. It makes a fine basis, however, for a profound ocular neurosis. If the ophthalmologist understands the nature of such eyes and their essential irritability, it is relatively easy · to put the patient at ease. Eyes like these are a nuisance, not a menace. They are an inherited handicap which must be overcome by perseverance and effort. Even with the best correction possible, they will never be any too comfortable, but no harm is done to the eyes in making them work. The fatigue, ocular and nervous, which results from this effort is relatively harmless when understood and philosophically endured.

It is the price to be paid for the things which one's ambition wishes to accomplish. The eyes will not be harmed in any way by such usage. Viewed in the light of this reasoning, when understood by the intelligent patient, the problem of the irritable anisometropic, astigmatic eyes is not difficult. In my experience, the reaction of the distressed persons with such eyes to this explanation is both grateful and satisfactory. Yet they constitute a rather large, troublesome and difficult group of patients in the average practice.

In connection with anisometropia and antimetropia, attention must be called to the work being done on aniseikonia, or difference in size of the images in the two eyes. This is an important work and should be watched. It is still in the experimental stage and has not yet been reduced to clinical application for those ophthalmologists engaged in office practice. Without doubt, in the near future the studies of this subject will have been thoroughly evaluated and put on a clinical basis.

There is also a definite and basic difference between hyperopic and myopic eyes. I formerly thought that the designations far sighted and near sighted were misnomers. I believe now, however, that they are good names. There is no doubt that myopic, or near-sighted, eyes are designed to do near work with ease and efficiency, whereas distance vision is more difficult for them. With hyperopic, or far-sighted, eyes the reverse is true. It is true that both near-sighted and far-sighted eyes can be made to do all kinds of work, but each excels in its own field. If they are called on to do work opposite from that for which they were designed, naturally more difficulty will be encountered.

The ability of the eyes to focus properly both for distance vision and for near vision is always a determining factor in ocular comfort. The status of the accommodative power and its relation to convergence constitute one of the most important problems in refraction. A knowledge of the state of the accommodation of each patient is absolutely necessary in the proper treatment of the condition. It determines not only the needs of the patient in regard to the strength of lens but by what methods he should be examined and what cytoplegic, if any, and how much should be used. One should measure the accommodative power in every case, the data thus obtained being indispensable in the evaluation of the eyes in question. As one does this one comes to rely more and more on this useful information. The normal accommodative values have been worked out carefully by Jackson[1] and Duane[2] and are

1. Jackson, Edward: The Amplitude of Accommodation at Different Periods of Life, and Its Relation to Eye-Strain, California State J. Med. 5:163-166 (July) 1907.

2. Duane, Alexander: Fuchs' Text-Book of Ophthalmology, ed. 8, Philadelphia, J. B. Lippincott Company, 1924, pp. 154 and 178-211.

available to use as a guide. To avoid repetition I refer the reader to two previous publications [3] which deal with this subject.

In discussing abnormal accommodative states, the following classification, modified after the arrangement by Howe,[4] may be useful:

1. Normal, near point of accommodation, average for age
2. Subnormal, near point of accommodation, below average for age
   (*a*) Paresis
      (1) Static or premature presbyopia
      (2) Dynamic or accommodative insufficiency
   (*b*) Paralysis
3. Spasm, relative excess of—near point of accommodation near normal
   (*a*) True spasm
   (*b*) Spasticity } associated with ametropia
4. Abnormally powerful or sustained—near point of accommodation close
   (*a*) Actual excessive, with ametropia
   (*b*) Actual excessive, with emmetropia

Of the conditions listed in this classification, I shall discuss briefly dynamic insufficiency, spasm and abnormally powerful accommodation. Certain instances of these three forms of accommodative disturbances are most interesting. Although such disorders are not common, when they do occur they cause marked difficulty for the patient and are likely to be overlooked by the ophthalmologist.

Subnormal accommodation manifests itself by inability to do near work or to sustain it in younger persons, those aged from 15 to 35 years of age. As persons of this age are supposed to have ample accommodative power, the condition will not be noted unless the accommodation is measured as a routine. The symptoms are those of accommodative asthenopia, and the accommodation will be found to be consistently below the average normal for the age. In these cases there seems to be a lack of that reserve potential accommodative power that usually is held in readiness to make near vision effective. Analysis of cases observed at the clinic showed that the condition was due to asthenia, debilitating conditions and focal infection, particularly dental sepsis. In some cases it seemed that the person naturally possessed a low accommodative power. Treatment was to relieve, if possible, the source of the trouble, after which the accommodation returned to normal in some cases; in other cases, however, the patient had to be given more plus lens for near work. For a fuller discussion of these cases I refer the reader to a previous publication.[3b]

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3. Prangen, A. DeH.: (*a*) What Constitutes Satisfactory Cycloplegia? Am. J. Ophth. **14**:665-671 (July) 1931; (*b*) Subnormal Accommodation, Arch. Ophth. **6**:906-918 (Dec.) 1931.

4. Howe, Lucien: The Muscles of the Eye, New York, G. P. Putnam's Sons, 1908, vol. 2, p. 26.

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    (a) Actual excessive, with ametropia
    (b) Actual excessive, with emmetropia

Of the conditions listed in this classification, I shall discuss briefly dynamic insufficiency, spasm and abnormally powerful accommodation. Certain instances of these three forms of accommodative disturbances are most interesting. Although such disorders are not common, when they do occur they cause marked difficulty for the patient and are likely to be overlooked by the ophthalmologist.

Subnormal accommodation manifests itself by inability to do near work or to sustain it in younger persons, those aged from 15 to 35 years of age. As persons of this age are supposed to have ample accommodative power, the condition will not be noted unless the accommodation is measured as a routine. The symptoms are those of accommodative asthenopia, and the accommodation will be found to be consistently below the average normal for the age. In these cases there seems to be a lack of that reserve potential accommodative power that usually is held in readiness to make near vision effective. Analysis of cases observed at the clinic showed that the condition was due to asthenia, debilitating conditions and focal infection, particularly dental sepsis. In some cases it seemed that the person naturally possessed a low accommodative power. Treatment was to relieve, if possible, the source of the trouble, after which the accommodation returned to normal in some cases; in other cases, however, the patient had to be given more plus lens for near work. For a fuller discussion of these cases I refer the reader to a previous publication.[3b]

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3. Prangen, A. DeH.: (a) What Constitutes Satisfactory Cycloplegia? Am. J. Ophth. **14**:665-671 (July) 1931; (b) Subnormal Accommodation, Arch. Ophth. **6**:906-918 (Dec.) 1931.

4. Howe, Lucien: The Muscles of the Eye, New York, G. P. Putnam's Sons, 1908, vol. 2, p. 26.

Spasm of accommodation is, I believe, closely related to dynamic subnormalcy. In a previous paper[5] I reported thirty cases of this condition. Like subnormal accommodation, it occurs in younger persons, those aged from 15 to 25 years. Characteristic of the difficulty is the apparent rejection by these eyes at the postcycloplegic test of proper correction of their hyperopia or a tendency to exaggerate their true correction for myopia as found with cycloplegia. At the postcycloplegic test or during a manifest refraction they will accept minus lenses when their error calls for plus lenses. The name spasm applied in these cases is misleading, as it perhaps implies excessive power; in reality, the condition seems to be one of irritability due to asthenia. These eyes are extremely uncomfortable unless relaxed by proper correcting lenses instead of a pseudomyopic correction. The symptoms are blurred distance vision, headaches, ocular discomfort and inability to use the eyes without fatigue. The accommodative near point in most cases is about normal for the age of the patient, although in a few cases it is intermittently too close. The primary cause of the phenomenon appears to be long-continued strain from attempting to adjust the ciliary mechanism to insufficient or improper correction, and this underlying cause is reinforced by secondary factors, such as functional disorders and focal infection. It is to be noted that the disturbing factor of anisometropia is almost always present. The nature of the trouble seems to be a cramp due to fatigue of an overworked and usually asthenic ciliary mechanism. In some respects a weak ciliary muscle and a spastic ciliary muscle are much alike. When subjected to undue fatigue the weak muscle gives up, whereas the spastic muscle continues to act in a spasmodic and inefficient manner. In either instance there is evidence of accommodative asthenia. The treatment of spasm is a proper correcting lens and a balance of the anisometropia. These eyes require a more accurate and balanced correction. At first the patient appears to refuse this correction, but usually within a few days or with the help of a little more homatropine he accepts the correction with gratitude. As a matter of fact, since I realized that the condition was one of irritability due to asthenia I have seldom used any more cycloplegic to relax the eyes to the new correction. The eyes adjust by themselves quickly. Of course one should bear in mind the age of the patient and not try to blur distance vision by too much spherical correction, as patients with this disorder are usually young. Also, anisometropic eyes are essentially irritable in spite of any correction. The patient should understand this and should not rush to get a new correction every time he has a relapse of symptoms, which occurs

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5. Prangen, A. DeH.: Spasm of Accommodation, with Report of Thirty Cases, Tr. Sect. Ophth., A. M. A., 1922, pp. 283-292.

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frequently. With stress, strain and fatigue the spasm is prone to recur. It will soon disappear again, however, with the continued wearing of the proper glasses and a return of physical and mental balance.

One occasionally observes instances of abnormally powerful or sustained accommodation which is the opposite of the conditions just described. It may occur in association with either ametropia or emmetropia. When seen in association with emmetropia, the condition is one of late presbyopia, the patients being 50 and even 60 years of age and not seeming to need reading glasses. One observes such instances every now and then. In association with ametropia one sees powerful accommodative mechanisms which seem to be able easily to carry marked hyperopic errors even at the ages of 38 to 43 years without the use of correcting lenses or possibly with the use of only low plus lenses for near work. Aside from interest in the unusual occurrence of such cases, the clinical point is the folly of trying to force glasses on these fortunate persons or to give them more than a mere minimum of correction for their needs. Their accommodation is such that they need little or no assistance.

Balance and imbalance of the extra-ocular muscles have always been a diffuse and difficult ophthalmologic problem. The diagnosis and treatment often seem obscure. I think it is possible to make this problem more simple by refining still further the usual conception of diagnosis. Too often diagnosis of the muscular condition stops with the statement that the patient has exophoria or esophoria, in distance vision or in near vision. No further effort is made to coordinate the relation of the phoria in distance vision to that found in near vision and the relation of the phoria to the powers of lateral and vertical duction.

There appear to be four steps in the completion of a refined diagnosis of a muscular condition: (1) the finding of a phoria (esophoria, exophoria or hyperphoria), (2) the determination of the degree of deviation for distance vision and for near vision and a comparison of these figures, (3) a study of the duction powers and (4) a study of the behavior of the individual muscles in their primary field of action. With these data at hand one is in the position to make an exact clinical diagnosis. The figures recorded are not of so much importance, I feel, as is a careful comparison of the relationships of the aforementioned data to one another. By putting together the information found from these four procedures the clinical diagnosis becomes apparent. All anomalies must be disturbances of convergence or divergence of the visual axes, either lateral or vertical. The table shows my arrangement of the points considered in this method of diagnosis. Following this line of reasoning, one can easily determine just what type of anomaly one has to deal with. It is sometimes true that deviations are the

same for distance vision and near vision. This is evidence of a compensated condition of long standing, which nature has rendered less troublesome by causing it to become continuous or constant. A little further study along the lines already mentioned will enable one eventually to figure out the true diagnosis. After the diagnosis has been made, the treatment, whether optical, medical or surgical, becomes much more clearly indicated. The type of treatment is naturally a matter of training, experience and personal preference. It is constantly changing and improving. I firmly believe, however, that a more refined and accurate differential diagnosis greatly simplifies the proper handling of imbalance.

An important point in the handling of imbalance is the constant association of anisometropia with conditions of this type. It may well be possible that uncorrected and improperly corrected errors of refraction, particularly when anisometropic, are the source of irritation and fatigue, which bring about the imbalance. If so, proper correction of

Excess and Insufficiency Under Various Conditions

Deviation Greater In	
Distance Vision	Near Vision

1. Exophoria
  a. Excess of divergence
	1. Exophoria
	  a. Insufficiency of convergence

2. Esophoria
  a. Insufficiency of divergence
	2. Esophoria
	  a. Excess of convergence

refractive errors is the first and most important step to be taken in the treatment of apparent muscular imbalance. Many times this is all that is necessary. If symptoms persist after a thorough trial of proper glasses, one must of necessity consider other measures, such as orthoptic exercise and surgical intervention. In a previous paper [6] I discussed the relationship between the differential diagnosis and the selection of operative procedures. I believe that a careful and refined differentiation of cases makes possible the selection of that combination of surgical intervention best fitted for each individual case. By this process of differentiation and selection it seems possible not only to obtain better cosmetic results but to arrive at the ideal of more normally functioning eyes.

This paper may be divided into two sections. In the preceding part some clinical aspects of refraction have been considered. The following observations pertain more to problems of technic and prescription.

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6. Prangen, A. DeH.: Surgery of the Rectus Muscles of the Eye: Selection of Operative Procedures by Differential Diagnosis, Tr. Am. Ophth. Soc. **32**:273-283, 1934.

PROBLEMS OF TECHNIC AND PRESCRIPTION

The patient who returns dissatisfied presents a difficult problem because he is likely to be handled improperly. The fact that he returns complaining or still uncomfortable implies that perhaps a mistake or an error in judgment has been made. It is natural for the examiner to resent this implication, and it is difficult to receive such a patient properly. The patient who returns dissatisfied is a challenge to the ability of the ophthalmologist. That he bothers to return is a compliment, in that this implies he still believes the ophthalmologist can help him and he is offering the ophthalmologist another chance if he will take it. Swallowing his pride, the ophthalmologist should receive him with good grace and disarm him by a pleasant willingness to try and set matters right. The ophthalmologist really should be glad that he returns, for often something needs adjustment. There may have been an error in the manufacture or adjustment of his glasses, or something in the prescription or findings may not check. After the ophthalmologist has received the patient decently it is relatively easy to correct anything found amiss, whereas an uncompromising attitude would have forced the patient to go elsewhere. These cases are not common, so that when they do occur one can well afford to recheck the findings completely in search of some unusual factor, error or overlooked detail. If after a careful recheck everything seems all right, the patient is usually willing to persist with the treatment or correction, for he has been pleasantly and tactfully received and thoroughly examined. The only remedy for an error or oversight is to recognize and correct it.

The indications for the use of a cycloplegic and its dose and sufficiency are of interest to all doing ophthalmologic refraction. A cycloplegic is used to relax the muscles of accommodation when accommodation is of an amount to interfere with the finding of the latent refractive error. When from age or illness the accommodative power has been reduced to 4 D. or less, cycloplegia is not, as a rule, employed, for such an accommodative power does not interfere with the finding of the total refractive error. Persons with such accommodative power usually have presbyopia and are more than 44 years of age. The dose and sufficiency of the cycloplegic seem to be indicated by these facts. In younger patients with more active accommodation, I believe that degree of cycloplegia is needed which will reduce the accommodative power to the level of presbyopia during the process of refracting the eyes. During the course of cycloplegia, as the accommodative level falls to less than 3 D. and approaches 2 D., the total refraction becomes easily manifested. In a large series of cases studied I found that a residual accommodation of 1.5, 1.7, 2 and 2.2 D. was satisfactory.

No additional latent error was found below these figures. As the accommodative power rose toward 3 and 4 D. the findings were uncertain and inaccurate. One can easily measure the residual accommodative power at the conclusion of a refraction in which cycloplegia was employed by adding a + 3.00 D. lens to the findings and measuring the accommodative range, the difference between the far point and the near point, in centimeters. This difference in centimeters, divided into 100, equals the range or remaining accommodative power, in diopters. This procedure enables one to determine the degree of cycloplegia obtained. One soon learns also, knowing the accommodative power of the individual patient, what dose of cycloplegic is needed to reduce the accommodative power to a satisfactory level in order to find the total refractive error without hindrance by the accommodation. The usage, dose and sufficiency of the cycloplegic, then, become a matter of measurement and adaptation to the individual patient rather than a matter of purposeless routine.

The therapeutic use of cycloplegia should also be kept in mind. Ophthalmologists often use it to allay ciliary irritability, spasm and fatigue. Eyes are put at rest and induced to accept the desired correction and to overcome bad ciliary habits resulting from insufficient or improper correction. A cycloplegic often is used in this way for patients who are in the late forties and early fifties, as well as for younger patients. I think that ophthalmologists are likely to forget these facts when confronted by persistently uncomfortable eyes.

The conception of astigmatism and its treatment is aided greatly by keeping in mind a picture of Sturm's astigmatic interval (shown in the figure). This interval is the space between the two principal focal points of an astigmatic lens system. The series of distorted images, occurring at different points in the interval, are representative of the images seen by an astigmatic eye when viewing an object. The clinical problem in astigmatism is to replace these images with the corrected image by obliterating the interval by means of a correcting lens, in other words, to bring the two principal focal points together. This would be a full correction of the astigmatism, an ideal. It is often necessary to give the eye a compromise image or partial correction for the astigmatism by partially obliterating the interval or bringing the focal points closer together. In initial attempts to correct higher degrees of astigmatism, the eye, being used to a distorted image or wide interval, may take more kindly to a gradual adjustment of the interval by partial or compromise corrections. There seems to be no logical reason why all eyes should be required to adjust to immediate full correction. The patient and the ophthalmologist often will be much happier when astigmatism is corrected gradually over a period of time. The tendency of American ophthalmologists, myself included,

mometer may indicate that all the astigmatism has not been uncovered);
(6) in a routine examination of the eyes, to help decide whether or not
a refraction should be done; (7) for the psychologic effect of the instru-
ment on the patient; (8) to differentiate between corneal and lenticular
astigmatism, and (9) to determine a doubtful axis in cases in which
even with astigmatism of 0.5 D. the axis varies from 90 to 180 degrees
in the cycloplegic and postcycloplegic tests.

It seems unnecessary to remark on the indispensability of the cross-
cylinder in determining the amount and the axis of astigmatism. Appar-
ently, however, the excellent articles of Jackson [8] and Crisp [9] are not
familiar to all. I urge a careful reading of these articles and an earnest
trial of this device. Once used, it will never be discarded. Although
I accept the method as described, there are several special points which
might be emphasized. For determination of the axis in ordinary errors
I prefer the weaker cross-cylinders, those of 0.12 and 0.25 D., so that
the vision will not be blurred too much. With the retinoscopic findings
in place, a preliminary axis is found. Subjectively the spherocylindric
relations are adjusted, and the best vision is obtained. As a final pro-
cedure the axis is checked again with the cross-cylinder. The axis often
will shift 5 degrees at this point, and I believe this final axis taken with
the best combination of lenses and keen· vision is the correct one,
although it may differ from the axis obtained at the preliminary exami-
nation. When the spherocylindric relationship has not been worked
out accurately, a wrong axis may be found. In using the cross-cylinder
for determining the amount of astigmatism, one is likely to obtain an
overcorrection or an undercorrection. This appears to be a phenomenon
of the interval of Sturm and is attributable to an improper relation
between the sphere and the cylinder. If the cross-cylinder appears to
lead one too far from the retinoscopic cylinder, the sphere should be
rechecked at each change indicated by the cross-cylinder. In other
words, the optical effect on both the sphere and the cylinder by the cross-
cylinder should be kept in mind when using it for strength. When it
is used for strength the cross-cylinder alters both the sphere and the
cylinder, and this alteration should be visualized. If the cylindric effect
alone is thought of and the cylinder is changed without the sphere being
checked, an increasingly distorted image will be produced, and the inter-
val of Sturm will be exaggerated instead of corrected. With these facts
in mind, the cross-cylinder can be employed satisfactorily in most cases
for finding the amount of astigmatism. There are, however, exceptions

8. Jackson, Edward: Principles Applied in Use of Cross Cylinders, Am. J.
Ophth. **12:**897-901 (Nov.) 1929.

9. Crisp, W. H.: The Cross-Cylinder Tests, Especially in Relation to the
Astigmatic Axis, Tr. Ophth. Soc. U. Kingdom **51:**495-514, 1931.

to the accuracy of the findings determined with the cross-cylinder for both the axis and the strength, and these findings must be checked by other methods.

In corrections for anisometropia and for antimetropia one should bear in mind the prismatic effect produced as the eyes look from distant to near objects. There occurs a shift of the optical center of the lens upward which produces the effect of a prism base up in hyperopia and that of a prism base down in myopia. This depends on the difference in the power of the two lenses in the vertical meridian. As the eyes are directed downward to read they are looking through the lenses 5 to 8 mm. below the optical centers of the lenses. If the lenses are of equal power in the vertical meridian, the prismatic effect is equal in each and is of no significance. This is not so, however, when the lenses are of unequal power vertically. The prismatic power produced in each lens is figured by the equation $A = P \times D$, in which A is the amount of prism produced, P is the power of the lens in the vertical meridian, and D is the distance in centimeters that the eye looks through the lens below its optical center. The difference in the prismatic power so produced in the two lenses represents the amount of vertical prism which the eye is asked to overcome while reading. To offset this, compensated lenses have to be ordered; for the nonpresbyope these consist of reading glasses with compensating prisms, and for the presbyope, of either reading glasses or special bifocal glasses with the addition of a prism. In spite of the theory, I do not think that compensated lenses are necessary in the majority of such cases, as the patients seem to adjust or tolerate their unequal corrections comfortably. One should bear this problem in mind, however, for the persistently uncomfortable anisometrope and give him the benefit of trial with compensated correction. Incidentally, I see no valid reason why in most cases of anisometropia and antimetropia the error should not be fully corrected eventually. In my experience the patients respond nicely to such correction.

One should consider also the effective power of lenses. The power of strong lenses, such as corrections for aphakia, varies considerably as their distance from the eye is changed. There may be a difference as great as 15 mm. anteroposteriorly in the position of lenses set in the trial frame and their position when they are worn by the patient in his glasses. The equation, $C = KSD^2$, is used to determine the change in power produced by moving a lens forward or backward in front of the eye. C is the amount of change produced in diopters; K is the constant, which is 0.001 when millimeters and diopters are used; S is the distance moved in millimeters, and D, the dioptric power of the lens. Thus the power of a 10 D. lens moved 10 mm. is altered 1 D. This problem, of course, is important only for lenses of higher power.

Ophthalmologists have had a long-standing aversion to the inclusion of prisms in prescriptions for glasses. It has been thought that their use would tend to exaggerate still further the muscular imbalance and cause the eyes to become dependent on them. Further, it has been thought that the amount of prismatic power necessary would increase as time went on. There is, of course, some truth in these ideas, and yet I believe prisms should be prescribed for wear when indicated. All ophthalmologists know that the wearing of correcting lenses for hyperopia and myopia makes the eyes dependent on them and that these corrections have to be increased as time goes on. Still I do not think that for these reasons correcting lenses should not be ordered. Increased visual efficiency and comfort demand these corrections. Likewise, it seems that a prismatic correction should be prescribed for the same reasons, namely, increased visual efficiency and comfort, whenever indicated. I have the feeling also that prisms are not prescribed more generally because of the lack of clear understanding of their action when they are placed in front of the eyes. Optically, one is taught that rays of light are deflected toward the base of a prism as they pass through it. Clinically, it is more useful to remember that the image of an object looked at through a prism is displaced toward the apex of the prism. Prisms are used for two purposes: (1) to displace or measure the displacement of the ocular images and (2) to make the eyes follow as far as possible displaced images. The first use is, of course, for the measurement or correction of deviations, diplopia or phorias. The second is for determining ductions or for exercise. Again one must understand the behavior of false images. These are found in the direction opposite to the deviation or the tendency to deviate of the eye because there has been a shift in the retinal pattern of the deviating eye without cerebral recognition of the fact. The brain continues to project the impulse received as though the eyes were directed normally. Therefore, in using prisms one either has or creates a false image, and prescription becomes simply a matter of knowing in which direction one wishes to displace the image and recognition of the fact that a prism displaces the image looked at toward its apex. When thus visualized, the use of prisms is very simple and also very useful for both diagnosis and prescribing.

In prescribing prisms I prefer to use a prism of the minimal degree with which single vision can be obtained. There is often considerable spread between the minimal and the maximal prism accepted. Other things being equal and the amount of prismatic power small, I prefer to place the prism before the nondominant eye. If the degree of the prism is more than 3, I prefer to divide it between the two eyes. To determine the minimal prismatic requirement, binocular fixation can be

fatigued by measuring the phoria slowly and bringing out the latent tendency to see double. Then before measuring ductions, the diplopia, if apparent, can be corrected by the minimal amount of prismatic power. Repeated tests of this character often help to find the amount of prismatic power necessary for single vision, for distant and for near objects. The amount of any such prism required must be added to or subtracted from the duction powers subsequently found. When vertical deviation differs for distance vision and for near vision (and it often does) one should not correct more than the lesser finding in glasses to be worn both for distance vision and for near vision. Otherwise, the eyes may be corrected for one distance and overcorrected or undercorrected for the other. If one wishes to give a different prismatic correction for distance vision and for near vision, either two pairs of glasses or special bifocal glasses are necessary. In cases of a combined lateral and vertical deviation the vertical deviation should perhaps be corrected first, although often both should be corrected, to give balance. Prisms seem to be most useful in the treatment of the insufficient convergence, divergence and vertical divergence and, conversely, least effective in the treatment of the excesses of these functions. Before prescribing prisms one should be sure that any existing ametropia has been corrected carefully and balanced vision has been obtained for both distant and near objects. Especially is this true in cases of anisometropia and antimetropia. Often when this has been done, apparent imbalances and symptoms disappear. If muscular imbalance and symptoms persist, however, the discriminating use of prisms seems to be next in line for consideration.

Abstracts from Current Literature

Anatomy and Embryology

PRESENCE OF GANGLION CELLS IN THE CILIARY NERVES OF THE ORBIT AND THEIR HISTOLOGIC SIGNIFICANCE. J. MAWAS, Bull. Soc. d'opht. de Paris, February 1936, p. 89.

The structure and rôle of the ganglion cells which form the compact mass of the ciliary ganglion have been well described. One may readily understand their function and final distribution. However, the presence of the ganglion cells in the ciliary nerves of the orbit needs histologic and physiologic elucidation. The problem of the accessory ganglions reported by Foesbeck, Svitzer, Antonnelli, Laffay, Fuchs and Axenfeld is not drawn into this question, but the author is concerned with the massed and isolated ganglion cells seen in the interior of the ciliary nerves. The classic textbook of Schieck and Brückner and that of Duke-Elder mention only their existence, but they were reported by Reichart in 1875 and, more recently, by Gallemaerts in 1899, André-Thomas in 1910, d'Erchia in 1895 and Cannieu in 1899. Mawas studied first the distribution of the ganglion cells with the nerve fibers. In the trigeminal and oculomotor nerves ganglion cells are noted in the terminal fillets of the nerves. The presence of the cells in the ciliary ganglion must be the origin of such cells in these nerve fibers. It is felt that such cells take the place of the sympathetic nerves to these parts.

L. L. MAYER.

Conjunctiva

THE HISTOPATHOLOGY OF PARINAUD'S CONJUNCTIVITIS. H. D. LAMB, Am. J. Ophth. 19: 571 (July) 1936.

Lamb gives a brief review of the literature on Parinaud's conjunctivitis, starting with the original article in 1889 and ending with a description of the pathologic changes noted by Verhoeff, who first discovered Leptothrix in the lesions. Two cases are reported. The following summary is given:

"The clinical reports and histologic findings with the presence of Verhoeff's leptothrix are described in two cases of Parinaud's conjunctivitis. In both cases, an eosinophilia of 4 percent was found."

W. S. REESE.

INTERESTING CASE OF SYPHILIS OF THE PALPEBRAL CONJUNCTIVA. PAULINA SATANOWSKY and P. KURLAT, Arch. de oftal. de Buenos Aires 11: 533 (Sept.) 1936.

This is the report of a case of an ulcerative disease of the palpebral conjunctiva, accompanied with swelling of the preauricular glands and

followed by secondary lesions of the throat. The patient gave a history of syphilis four years before. The authors, after a full discussion of the different elements in the case, express the opinion that the condition was a primary lesion (due to reinfection after a prior cure).

<div align="right">C. E. FINLAY.</div>

A CASE OF PEMPHIGUS OF THE CONJUNCTIVA. D. M. BAKHIR, Sovet. vestnik oftal. 9: 920, 1936.

This case is reported because of the rarity of the condition.

A young healthy person had pemphigus of the urethra, with formation of a stricture which took a malignant course, as the artificially made fistula also became stenosed. There was pemphigus of the conjunctiva of the lower fornices of each eye, with scar formation. The mucous membrane of the mouth (the palate) was also affected.

<div align="right">O. SITCHEVSKA.</div>

Congenital Anomalies

OCULAR NEVUS. D. ARGÜELLO and C. VON GROLMANN, Arch. de oftal. de Buenos Aires 11: 417 (July) 1936.

Argüello and von Grolmann first give the results of their studies of melanosis and the origin of pigments in the body—(1) hematogenous, (2) cellular (lipochromes and melanin), (3) metallic and (4) rhodopsin. Melanin seems to originate in a biologic adaptation to radiant energy and is especially abundant in tropical races. The authors discuss the different racial types and the distribution of melanin in different ocular tissues and report the case of an Argentine with lesions of the skin of the lids and temple, deposits of melanin in the conjunctiva, sclera and iris and a semilunar area of pigmentation of the disk, without any depreciation of vision.

<div align="right">C. E. FINLAY.</div>

MULTIPLE PIGMENTED CONGENITAL CYSTS OF THE IRIS. J. A. SENÁ, Arch. de oftal. de Buenos Aires 11: 462 (Aug.) 1936.

After giving a detailed classification of cysts of the iris according to their origin, with abundant bibliographic references, Sená stresses the production of spontaneous interepithelial cysts due to nonfusion of the two layers of the epithelium of the iris and reports a case of multiple cysts of this nature near the pupillary margin in each eye. A stereophotograph and a colored drawing illustrating the condition are included.

<div align="right">C. E. FINLAY.</div>

THE OPTIC VESICLE, COLOBOMAS OF THE FUNDUS OCULI AND OTHER MALFORMATIONS. C. S. DAMEL and E. ADROGUÉ, Arch. de oftal. de Buenos Aires 11: 473 (Aug.) 1936.

This is a lengthy paper, which does not lend itself to abstracting, in which the authors review at length the literature on the subject given in

the title. They discuss different points of view, classifications and the mode of production of the malformations. The article is illustrated with numerous cuts and photomicrographs, many of original preparations.

C. E. FINLAY.

Cornea and Sclera

THE ASSOCIATION OF DENDRITIC ULCER OF THE CORNEA AND OF SUPERFICIAL PUNCTATE KERATITIS WITH HERPES FACIALIS. H. NEAME, Brit. J. Ophth. 21: 298 (June) 1937.

The first patient, a man aged 49, when first seen had a sharp febrile attack, a marked herpetiform eruption of the face and fairly extensive dendritic ulceration of the cornea in two areas, one small and the other large. The second patient was a man aged 29 with pulmonary tuberculosis, which had been treated by artificial pneumothorax. Associated with the condition of the chest there was herpes facialis. Later there was a dendritic ulcer of the cornea, with two spots that were classed as nummular or macular keratitis. The third patient was a man aged 28 with typical superficial punctate keratitis, with two small dendritic ulcers near the margin of the cornea. The condition was associated with herpes facialis.

Neame discusses the pathogenesis, etiology, symptoms and signs of the superficial type of corneal ulcers.

The article has three illustrations.

W. ZENTMAYER.

SYPHILITIC LESIONS OF THE CORNEA AND THE CONJUNCTIVA IN A NEW-BORN INFANT. NICOLAS, P. BONNET, GONNET, BANSILLON and COLRAT, Bull. Soc. d'opht. de Paris, February 1936, p. 97.

In the corneal ulcers of a premature infant the spirochete was observed. The Wassermann reaction of the blood had been negative. In the first few days the infant showed conjunctivitis of each eye, which was more marked in the left eye, with edema of the lids, chemosis and staphylococcic suppuration. Ulcers of the corneas were present, and, in addition, interstitial infiltration with vascularization. Consultation with Professor Nicolas, of the department of diseases of the skin, resulted in a diagnosis of syphilis, and the treponema was observed in corneal scrapings. The question of hereditary syphilis is discussed. The liver and the spleen were not enlarged, and there were no buccal lesions. No chancre was found in the examination of the passages. An isolated enlarged gland was found in the maxillary angle. This was very hard. General treatment, consisting of mercury rubs, was given. Recovery was immediate. The mode of contamination remains obscure. The Wassermann reaction of both parents was negative.

L. L. MAYER.

CORNEAL ULCERS OBSERVED AFTER CERTAIN INJURIES IN PREGLAUCO-MATOUS EYES. A. VON NAGY, Klin. Monatsbl. f. Augenh. 97: 523 (Oct.) 1936.

Nagy reports the case histories of three patients with apparently irrelevant corneal injuries followed by obstinate corneal ulcers and

glaucoma later on. These patients and others with a similar course of their injuries were of advanced age, and, considering their injuries slight, they neglected them for a week or longer. The injuries of the three patients in question were caused by a splinter of rock, a chip of wood and a darning needle, respectively. The intra-ocular tension was raised on admission to the polyclinic. Nagy thinks that these eyes were predisposed to glaucoma, which became manifest probably as a result of dilatation of the ciliary vessels by means of irritation of the sympathetic nerves. A number of publications in point are quoted, and the result of Ehrlich's experiment with fluorescein, applied in these three cases, is described. The author arrives at the following conclusions: 1. Mydriatics must be used with caution in cases of traumatic corneal ulcers in patients of advanced age. 2. Posterior sclerotomy should be done at an early date. 3. An attack of glaucoma occurring in one of these patients later on is clinical proof for the correctness of the view arrived at regarding the glaucomatous diathesis. 4. Correct evaluation of the complaints may be of preventive importance for both eyes.

<div align="right">K. L. Stoll.</div>

CALCEMIA AND CALCIUM THERAPY IN SO-CALLED ECZEMATOUS BLEPHAROKERATOCONJUNCTIVITIS. D. LĂZĂRESCU and E. DAMIAN, Klin. Monatsbl. f. Augenh. 97: 626 (Nov.) 1936.

Lăzărescu and Damian discuss the relation of tuberculosis to the type of blepharokeratoconjunctivitis which is inadequately called "eczematous." This disease occurs frequently in persons with latent tuberculosis, although the bacillus of Koch could never be found in phlyctenules. The research on calcium deficiency as a cause of edema and other disturbances is evaluated, and the bibliography in point is cited. Data are given relative to the calcium content in the protoplasm of the cell, the vitreous, the blood serum and elsewhere in normal conditions and pathologic conditions and in pregnancy. Among the divers actions of calcium given therapeutically, its sedative action in some ocular diseases is referred to as described by Georgiana Theobald.

The authors examined the calcium content of seventy persons, using the method of Kramer and Tisdall. These patients suffered from divers ocular diseases, forty from fulminant eczematous keratoconjunctivitis or blepharokeratoconjunctivitis, which was combined with phlyctenules in most of them. Tracheobronchial adenopathy was roentgenologically found in nine children who, however, had no pulmonary lesions. The blood for the tests was taken after the first breakfast. The calcium content of the normal persons varied between 92.21 and 112 mg. per thousand cubic centimeters of serum, with an average of 100 mg., except for pregnant women, for whom the average was 88.8 mg. In seventeen patients with eczematous conjunctivitis the values varied between 83 and 89 mg. These patients received local treatment and 10 cc. of calcium intravenously every forty-eight hours. The calcium content increased with the treatment. The duration of the treatment was seventeen days, and no untoward effects of the injections were noted. Calcium gluconate and calcium thiosulfate administered intramuscularly or intravenously not only promptly reduce photophobia,

spasms and edema but also accelerate recovery, with reduction of hospitalization to one half, and incidentally improve the general physical condition of the patient. Intravenous injections act more promptly, intramuscular injections more protractedly. The settling time of the erythrocytes, which is accelerated in eczematous keratoconjunctivitis, is retarded by this treatment, as a result of general physical improvement.

K. L. STOLL.

Experimental Pathology

THE EFFECT OF GAMMA RAYS ON CELL DIVISION IN THE DEVELOPING RAT RETINA. K. TANSLEY, F. G. SPEAR and A. GLÜCKSMAN, Brit. J. Ophth. **21**: 273 (June) 1937.

The object of the experiments undertaken by the authors was:

1. To determine the precise effect of gamma irradiation on the division of cells in mammalian tissue.

2. To compare the results with those previously obtained with chick tissue.

3. To determine quantitatively the alteration in division of cells and the production of cellular degeneration after different doses of radiation.

4. To investigate the relationship, if any exists, between the mitotic changes and the degeneration which follows irradiation.

The following summary is given:

"1. A method of exposing the developing rat eye to fairly accurate doses of gamma radiation is described.

"2. It has been found that the normal 50:100:50 value for the prophase:metaphase:telephase ratio is altered as a result of irradiation. The distortion of the ratio varies with the size of the dose and the time-interval between irradiation and examination of material.

"3. A relationship has been established between alteration in cell division and the occurrence of degeneration among undifferentiated cells.

"4. The results show that cellular degeneration can be produced by very small doses of gamma rays and that this degeneration is accounted for by an effect upon cells which are about to divide which unfits them for the ordeal of mitosis.

"5. The quantitative results obtained from this mammalian tissue closely resemble those already provided by experiments on avian fibroblasts cultivated *in vitro*.

"6. A statistical analysis has been made of the individual variation of mitotic counts in different rats, both irradiated and unirradiated."

W. ZENTMAYER.

General

THE INTRA-OCULAR TENSION AND PREGNANCY. E. DE GRÓSZ, Ann. d'ocul. **174**: 167 (March) 1937.

The intra-ocular tension depends on a number of factors: the pressure of the intra-ocular vessels, the permeability of these vessels, swell-

ing of the vitreous and filtration of the aqueous. The regulation of these functions is governed by the vegetative centers, which are under hormonal influence.

Among 50 pregnant women examined, Hertel and Imre found the following results: In 42 the tension was diminished; this condition existed particularly in those who had acromegaly. In 2 the tension was normal, and in 6 it was increased.

De Grósz himself observed 105 patients, and in this group he made 151 examinations. The results are set forth in a number of tables and charts. A bibliography is given.

S. H. McKee.

New Method of Anesthesia and of Elective Paralysis of Various Nerves and Muscles of the Orbit. C. Dejean, Arch. d'opht. 53: 32 (Jan.) 1936.

Dejean points out as an objection to massive orbital injection the fact that the eye is immobilized and turned up, which is a poor position for most operations. The purpose of his study has been to devise a means of paralyzing one muscle or one group of muscles so that the eye will be turned in the direction desired. He first describes in detail a method of injecting the infra-orbital nerve in its canal. For obtaining paralysis of the superior rectus muscle a 5 to 6 cm. needle is used to puncture the skin from 3 to 4 mm. below the external angle of the lids; this passes round the globe and is carried backward and upward in the direction of the parietal boss of the opposite side. The injection is made when the needle has entered a depth of from 3 to 5 cm. The relations of the other rectus muscles and the nerves are described, and the procedures necessary to affect them are given. The conditions necessary for their elective paralysis are (1) a small amount of the fluid for injection, (2) injection back of the fibrous sheath of the muscle and (3) injection as nearly as possible where the nerve enters the muscle. These relationships are all given in detail.

S. B. Marlow.

General Diseases

Dental Focal Infection in Ophthalmology. C. Charlin and L. Gomez V., Arch. de oftal. de Buenos Aires 11: 251 (May) 1936.

This paper is based on the findings in fifty cases taken from Charlin's clinical records and from the literature, in all of which the ocular lesion was resistant to the usual therapeutic measures and in which spectacular improvement followed destruction of the dental focus.

The ocular lesions fell under the following headings: conjunctivitis, three cases; keratitis (vesicular, three cases; ulcerative, three cases; parenchymatous, two cases) eight cases; karato-iritis, one case; episcleritis, one case; iritis, ten cases; iridocyclitis, four cases; cyclitis, two cases; solitary retinal hypertension, one case; retinal angiospasm, one case; neuroretinitis, four cases; retrobulbar neuritis, three cases; oculomotor paralysis, five cases; orbital cellulitis, one case; nasal syndrome, two cases, and glaucoma, one case.

As a rule, the ocular disease is irregular in its course, with abrupt exacerbations and remissions, and the clinical and laboratory findings are negative.

The lesion was unilateral in 94 per cent of the cases.

Details of the ocular lesions are noted.

As regards the provocative dental lesion, in thirty-four of the fifty cases it affected a single tooth; in forty-seven cases it was painless, and almost invariably the affected tooth was an obturated tooth with 4 degree caries; in most cases the affected tooth had multiple roots. In most cases in which a single root was affected the tooth was in the upper maxilla of the same side as the ocular lesion.

The dental focus is produced by an anaerobic micro-organism; the ocular lesion contains no germs.

A detailed abstract of the fifty cases closes the paper.

C. E. FINLAY.

General Pathology

THE CAPILLARIES IN THE NAIL BED IN DISEASES OF THE EYE. S. MIELKE, Arch. f. Augenh. 110: 236, 1936.

In a large number of cases of diseases of the eye the capillaries of the nail bed were examined with the microscope. Mielke came to the following conclusions:

1. Capillary hemorrhages in the nail bed, as distinguished by Marchesani as an important sign of vascular disease, are frequent.

2. The frequency of these hemorrhages does not run parallel with age, even though arteriosclerosis plays a rôle.

3. Such hemorrhages were found more frequently in persons with myopia, especially myopia with degenerative changes, and in young patients with paralysis of ocular muscles in whom neurologic examination showed no signs of tabes or multiple sclerosis.

4. In cases of inflammation of the uvea there was no special frequency of hemorrhages in the nail bed, and the same was true in cases of glaucoma. The investigations in the patients with glaucoma were not sufficiently extensive to enable one to form any definite conclusions.

F. H. ADLER.

Glaucoma

VARIATIONS IN A FILTRATING CICATRIX IN ONE EYE IN THE COURSE OF GLAUCOMA OF THE UNOPERATED OR FELLOW EYE. J. SÉDAN, Bull. Soc. d'opht. de Paris, February 1936, p. 91.

A curious variation was seen in an eye which had been successfully operated on for glaucoma by the Lagrange technic in 1931 when chronic simple glaucoma developed in the fellow eye in 1935. The right eye, which had been operated on in 1931, had manifested glaucoma after hypertension and uremia. The operative scar, 2 mm. in diameter, showed no change, and the tension of the eye remained 20 mm. of mercury. In May 1935 the patient had profound anemia, the onset of

which was followed by violent pain in the left eye. Acute glaucoma supervened, which was relieved by miotics, with recovery of the normal tension and normal visual acuity. Sédan noted that the filtrating scar of the right eye increased in size fourfold and presented edema of the upper bulbar conjunctiva. He was not interested in the tension, fields or vision of this eye. On relief of the acute condition of the left eye the signs in the right eye subsided. The hypothesis of a general reaction with specific toxic effects of a sympathetic nature is strengthened by this phenomenon.

L. L. MAYER.

Lens

INTRACAPSULAR EXTRACTION OF CATARACT BY THE HUNGARIAN METHOD. G. PARTE, Ann. d'ocul. 174: 248 (April) 1937.

Parte's intention is not to show the advantages of extraction of cataract in its capsule over the classic method of extracapsular extraction but to draw the attention of ophthalmologists to the operative procedure of intracapsular extraction by a forceps, which is simpler than the procedure generally adopted and which, referring to Emil de Grósz, of Budapest, he calls the Hungarian method.

After a description of the preparation, the retrobulbar injection and a stitch for security, the incision and the iridectomy (the pupil having been previously dilated) are described. The capsule of the lens is grasped near the inferior extremity of the vertical meridian. The zonule of Zinn is then progressively broken, and the lens is extracted by executing an interior version in such a fashion that the inferior part of the equator is brought to the corneal surface. Multiple sutures are used.

Parte has reported this method in the hope that ophthalmologists who have not already done extraction of the lens in the capsule will try it.

S. H. McKEE.

LENTICULAR ANTIGEN THERAPY. I. BORSOTTI, Ann. di ottal. e clin. ocul. 64: 744 (Nov.) 1936.

The literature on lens protein, including the clinical reports of Davis, Marquez and others, is reviewed. Borsotti believes that the reports of Davis on the use of lens antigen in incipient cataract are without any sound basis, but that the work on the absorption of cortex after discission in patients given lens antigen must be considered more seriously. In attempting to repeat the work of Biffis and Quaglio he found that after discission in guinea-pigs the results did not permit exact observation, since the amount of cortex in the anterior chamber varied. Instead, he injected an emulsion of beef and rabbit lens so as to fill the anterior chamber after removal of the aqueous. Each of fifty rabbits was given such an injection, and half of them were also each given a subcutaneous injection of beef lens either previous to or after the injection into the anterior chamber. The animals were divided into groups according to various conditions of immunization. Those animals which received subcutaneous injections after the injections into the anterior chamber showed no difference in speed of absorption from the controls. Some of

those which had received the preliminary course of injections showed more rapid absorption (eleven animals). Phenomena of sensitization were seen in this group, however, as shown by a marked inflammatory reaction when injection was made into the anterior chamber. While the subsequent injection did not hasten absorption, it apparently lessened the inflammatory reaction due to the presence of lens material in the anterior chamber. Borsotti concludes that his work does not encourage the use of lens antigen to hasten absorption of cortex but indicates that this may involve risk of provoking an anaphylactic reaction at the time of operation. He believes passive immunization with an immune serum would be free from this danger and should be investigated.

A bibliography is included.

S. R. Gifford.

PROLAPSE OF VITREOUS AND GLAUCOMA AFTER INTRACAPSULAR EXTRACTION OF CATARACT. R. F. PEREIRA, Arch. de oftal. de Buenos Aires **11**: 409 (July) 1936.

After reference to a former paper on prolapse of vitreous followed by glaucoma after discission of a secondary cataract, Pereira reports two cases in which there was a similar complication after intracapsular extraction of cataract. In one case the condition followed a violent physical effort, and in the other it occurred spontaneously after pupillary dilatation.

C. E. Finlay.

Lids

AMYLOIDOSIS OF THE LID. O. I. RYBNIKOVA, Sovet. vestnik oftal. **9**: 911, 1936.

Up to 1928 there were recorded in European and American literature only one hundred and thirty-three cases of amyloidosis of the lid.

A man aged 63 complained of gradually increasing swelling of the right upper lid. Examination revealed marked ptosis of the right eye; the lid was thickened and had a normal skin with smooth folds, and the margin of the lid was S shaped. A tumor about 1 cm. wide could be palpated in the lid; it was elastic, painless and movable and had a rough surface. It was impossible to evert the lid. There was a small round tumor of the scleral conjunctiva at the inner angle. There was present trachoma of stage 3 with pannus of the cornea. A biopsy confirmed the clinical diagnosis of amyloidosis. Extirpation of the upper tarsus with consequent transplantation of the mucous membrane of the lid was done. The ptosis disappeared shortly after the operation, and the lid became normal.

A detailed description of the pathologic process and of the various staining methods used is given. The outstanding feature was the abundance of homogeneous large and small masses with homogeneous fibers between them.

The clinical, pathologic and physiochemical properties of amyloid are described. Various theories of the etiology of amyloidosis are discussed. The association of deposits of amyloid with trachoma and the predisposition of chronically irritated tissues to amyloidosis is noted.

O. Sitchevska.

Neurology

GENICULATE NEURALGIA (NEURALGIA OF THE NERVUS FACIALIS): A FURTHER CONTRIBUTION TO THE SENSORY SYSTEM OF THE FACIAL NERVE AND ITS NEURALGIC CONDITIONS. J. RAMSAY HUNT, Arch. Neurol. & Psychiat. 37: 253 (Feb.) 1937.

Although this article is primarily concerned with neuralgia arising from the geniculate ganglion, nevertheless the discussion of the anatomic features of the sensory and sympathetic nerves of the face and their relation to deep orbital pain should be of interest to all ophthalmologists. There is a discussion of sphenopalatine ganglion neurosis (Sluder's syndrome), the pain of which is localized in the root of the nose, in and about the eye and in the upper part of the face and the upper teeth and sometimes also in the lower jaw and the lower teeth, in the temple and about the zygoma, the ear, the mastoid, the occiput and the neck, the hand and even the finger tips.

Hunt summarizes the neuralgic disorders of the various cranial nerves as follows:

1. True trigeminal neuralgia, which is distributed in one or more branches of the trifacial nerve and in which the pain is localized in the more superficial structures of the face and intra-oral region. This is classic prosopalgia or trifacial neuralgia. In cases of neuralgia of the third division of the fifth nerve there is often associated otalgia.

2. Geniculate neuralgia, which involves the deeper structures of the face. This is characterized by pain in the deep posterior orbital, palatal and nasal regions, with painful pressure sensation in the face. This is geniculate deep prosopalgia, and with it there is associated geniculate otalgia.

3. Glossopharyngeal neuralgia, which is characterized by neuralgic pains in the distribution of the glossopharyngeal nerve at the base of the tongue and the adjacent regions of the throat and by associated otalgia.

4. Superior laryngeal neuralgia, of vagal origin, in which the pains are localized in the region of the larynx, with associated otalgia.

All these various forms are accessible to surgical intervention by the cranial method of approach through the posterior fossa, which exposes the fifth, seventh, ninth and tenth cranial nerves. If this procedure is carried out with the use of local anesthesia, it is possible by touching any one of the nerves to reproduce the neuralgic pain, thus confirming the clinical diagnosis, which is often involved and difficult.

An excellent series of references is included.

R. IRVINE.

CERTAIN REACTIONS OF THE OCULONASAL NERVES. L. HALBRON, Ann. d'ocul. 174: 145 (March) 1937.

The related reactions of the eye and the nose, both physiologic and pathologic, have long been known. Simultaneous stimulation of the organovegetative system of the orbit and that of the nasal fossae causes sensory and vasomotor syndromes which are no more pituitary of ocular origin than orbital of nasal origin. Halbron purposely does not discuss suppurative sinusitis and the orbital complications.

In the section on the clinical study of oculonasal nervous reactions the author discusses (*a*) the syndrome of the nasal nerve, reporting two cases observed by him in which the symptoms of pain, ocular disturbances and acute rhinitis occurred, and (*b*) the syndrome of the sphenopalatine ganglion, the symptoms of which are pain and involvement of sympathetic nerves.

In the section on diagnosis of the syndrome of the nasal nerve and that of the sphenopalatine ganglion he discusses these syndromes and then the clinical diagnosis of the two syndromes in relation to various types of facial neuralgia. The last topic is subdivided into neuralgia of facial nerves, pain in the ophthalmic zone and neuralgia of facial sympathetic nerves.

Treatment of these syndromes often proves their spasmodicity and is therefore of diagnostic value. Local treatment recommended is the introduction of cocaine into the nostril; general treatment consists in soothing the nervous system and attempting to eliminate intoxications or endocrine disturbances.

                                                              S. H. McKee

Operations

THE FLUORESCENT LAMP FOR CATARACT SURGERY. H. R. HILDRETH,
    Am. J. Ophth. **19:** 770 (Sept.) 1936.

Hildreth describes the fluorescent lamp and enumerates its uses as a therapeutic and diagnostic agent and for photography of the eye and surgery of the lens. He emphasizes the latter.

                                                              W. S. REESE.

MARGINAL CUTANEOUS BLEPHAROPLASTY IN PALPEBRAL REPAIRS.
    L. DUPUY-DUTEMPS, Ann. d'ocul. **174:** 312 (May) 1937.

In cutaneous blepharoplasty, when the loss of substance is not extensive, the edge-to-edge tarsorraphy usually employed gives sufficient exposure on the surface to allow a good result. However, in cases in which there is strong fibrous retraction and the lid has to be reconstructed in the greater part of its width, this method is insufficient. The opposite palpebral border in these circumstances has been a good deal lowered and usually has an inflamed surface. The result of an attempted blepharoplasty under these circumstances remains imperfect, and although the lagophthalmus and ectropion may be lessened, they still persist. This insufficiency is met with especially in cases in which extensive repair of the lower lid is required and particularly if the eye is missing or has become atrophic.

Dupuy-Dutemps describes in detail, supplying four illustrations, an operation which he considers satisfactory, in the conditions already described.

                                                              S. H. McKee.

FIRST SERIES OF PARTIAL PENETRATING CORNEAL TRANSPLANTATION.
    I. F. KOPP, Sovet. vestnik oftal. **10:** 347, 1937.

Kopp performed thirty corneal transplantations from March 1934 to March 1937 after von Hippel's, Elschnig's and Filatov's method. The first eleven patients whose cases are reported here were followed

from twelve to eighteen months. In five of these the transplant took and was transparent, and vision was improved from counting of fingers to 0.1 (three cases), 0.2 and 0.4, respectively.

A detailed analysis of the cases, a description of the postoperative course and the results of histologic examination are given. Successful transplantation was observed, first, in those cases of leukoma in which there was a sufficient amount of sound corneal elements spread over a wide operative field, second, in those cases in which the anterior chamber was present and, third, in those cases in which the process which caused the corneal opacity was completed, particularly in cases of trachomatous pannus. The injury of the endothelium and the formation of folds in Descemet's membrane of the transplant favor the production of fibrinous exudate, with resultant vascularization and connective tissue scarring. Kopp injects epinephrine hydrochloride subconjunctivally forty minutes before the operation in order to keep the pupil dilated. Traumatization of the lens occurred only once. Kopp considers Filatov's hand trephine sufficient and effective for the operation. He recommends the wide use of the cadaver's cornea because the transplant is taken from a healthy eye and because the material can be obtained whenever it is needed.

Corneal transplantation was performed on forty-five rabbits previous to the keratoplasty performed on human beings.

<div align="right">O. Sitchevska.</div>

Orbit, Eyeball and Accessory Sinuses

Unusually Large Mucocele of the Frontal Sinus, with Severe Visual Disturbances and Recovery. F. Csillag, Klin. Monatsbl. f. Augenh. 97: 663 (Nov.) 1936.

The right eye of a woman aged 52 was swollen for about five months prior to observation by the author. The attending general practitioner had tried to empty the tumor by insertion of a cannula into the upper lid. Preceding grip, syphilis and tuberculosis were excluded. The right palpebral fissure was narrowed and displaced downward and temporally; the upper lid showed ptosis and was immovable, cyanotic and crossed by distended blood vessels. No tenderness on touch existed. A massive. tight but barely fluctuating tumor the size of a walnut was seen below the upper lid. It arose from the frontal bone, causing exophthalmos. The upper orbital margin was not palpable. The eyeball was flattened from above. The disk was edematous and prominent; the retinal veins were slightly distended and tortuous, and retinal hemorrhages were absent.

A roentgenogram revealed the upper margin of the orbit greatly deviated upward and the entrance of the orbit and the frontal sinus generally widened, while the periosteum of the temporal upper margin was thickened. An incision was made along the eyebrow; the lid was pulled downward, and the tumor was removed; it burst during extirpation. The mucocele extended backward toward the optic foramen. diverting the optic nerve; the osseous wall of the orbit and an area of the dura mater were eroded. Nevertheless, prompt recovery followed. and vision of 5/10 of normal was regained, with a prospect of further improvement.

<div align="right">K. L. Stoll.</div>

Pharmacology

DOES ERGOTAMINE TARTRATE CAUSE PARALYSIS OF THE SYMPATHETIC NERVE OF THE IRIS? E. FROMMEL and D. ZIMMET, Ann. d'ocul. **174**: 178 (March) 1937.

It is known that ergotamine tartrate acts clinically as if it paralyzed the sympathetic nerves; there is not a neurologist or a cardiologist, who, experimenting with the action of this drug, is not surprised by the quieting qualities of this medicine on the sympathetic nervous system. Frommel and Zimmet have shown that the tachycardia of rabbits which have for many days been given injections of thyroxin or of an estrogenic substance was influenced immediately by ergotamine injected intravenously.

The details of the experiments of the authors are described. They conclude from their studies that the miosis provoked by ergotamine tartrate is not only of a paralytic order, because excitation of the sympathetic nerves produces also mydriasis. The drug does not totally paralyze the ocular sympathetic nerves. Ergotamine tartrate, in other words, does not inhibit the action of epinephrine on the sphincter of the iris, as is the case with the arterial pressure. S. H. McKEE.

Physiology

THE SPHENOPALATINE GANGLION AND THE EYE. DUBOIS-POULSEN, Ann. d'ocul. **174**: 217 (April) 1937.

Certain interested advertisers have brought into style reflex therapy, especially that related to the nasal mucosa. It is presented as a miraculous method and a cure-all. In 1925 Halphen showed that these treatments were directed to the sphenopalatine ganglion.

This ganglion possesses numerous connections with the eye, and it is natural to think that the therapeutic successes obtained in the field of general medicine might be repeated in that of ophthalmology.

Dubois-Poulsen reports on the anatomic, physiologic and pathologic aspects of his studies. Under the heading Physiologic Aspects come the following: I. Secretory Activity: Tearing with Stenosis of the Lacrimal Tract. II. Trophic and Vasomotor Disturbance: Lesions of the Anterior Segment of the Eye. III. The Rôle of Sensitivity: Facial Allergies, Glaucoma, Atrophy of the Optic Nerve and Diverse Lesions.

Much pain is referred to the region of the sphenopalatine ganglion, and for this reason study of the ganglion and the relief of pain by anesthesia merits attention. A bibliography is appended.

S. H. McKEE.

Retina and Optic Nerve

INFANTILE AND CONGENITAL RETINAL FOLD. A. TILLEMA, Brit. J. Ophth. **21**: 94 (Feb.) 1937.

A slight divergent squint had developed in a child 3½ years old, and the eye looked strange. The lens was turbid in its deeper layers, especially at the center. Behind the lens, temporally and below, a fairly well defined mass was seen against the retina. The eye was

removed. Microscopic examination showed a few posterior synechiae. The lens was liquefied and vacuolated in the anterior cortex. Delicate fibers of connective tissue were attached to the posterior surface and ran toward the ora serrata. The retina was completely detached. It showed two folds, one extending from the optic papilla toward the pars plana of the ciliary body, downward and slightly outward, and a second just behind the ora serrata, extending in a circular direction. The crests of both folds were attached to strands of connective tissue. All these fibers joined behind the lens in a fairly vascular mass of connective tissue. The optic papilla was 2 mm. in front of the retina. Many of the retinal rods and cones were missing. Many small spaces were filled with a homogeneous coagulation. Scattered leukocytes were present. The retinal veins were surrounded by closely packed lymphocytes. Close to the large fold the retina exhibited disorganization and a hole. In this region the inflammatory changes were most marked. The optic nerve traversed the subretinal space for a distance of 2 mm. From the optic nerve a strong fibrous band continued forward. It contained many fibroblasts. The retina and connective tissue membrane contained scattered polymorphonuclear leukocytes. The central artery of the retina divided into several branches close to the optic papilla. No remnants of a hyaloid vessel were observed.

The inflammatory changes were confined almost entirely to the retina and vitreous, and for this reason it is not difficult to reconstruct the pathologic process. Retinitis, probably metastatic, was followed by abscess formation in the vitreous. This abscess subsided and was organized. Fibrous tissue then contracted and pulled every structure to which it was attached toward the center of the eyeball. Thus the retina, optic nerve and choroid were displaced.

Tillema concludes that in his case the anomaly was caused by inflammation, probably retinitis; in Weve's eighth case it was probably caused by cyclitis of the pars plana of the ciliary body. It is suggested that in Weve's first, fourth, sixth and seventh cases the condition was related to high myopia. For all other cases an inhibition of normal development of unknown origin must be accepted; for the group a familial occurrence has not been accepted.

The article is illustrated.

W. Zentmayer.

DIFFUSE DETACHMENT OF THE RETINA IN A SCROFULOUS MYOPE. BUJADOUX, Bull. Soc. d'opht. de Paris, February 1936, p. 100.

A woman of 38 years consulted Bujadoux because of high myopia. Visual acuity of the left eye was brought to 1.0 with a —7 D. sphere Vision of the right eye was barely 1/2 with the same correction. Examination of the fundus revealed a large white lesion behind the retina in the vicinity of the right macular area, surrounded with a transparent zone. The patient was being treated for Pott's disease by the orthopedic department. Subconjunctival injections of mercury were of no avail. During this treatment new lesions of similar nature appeared in the temporal portion of the retina. Gradually the entire temporal portion of the retina, including the macular area, became detached. Treatment with the methylic antigen resulted in maintenance of vision of 1/2. The detachment persisted but remained the same. The patient was

presented to obtain advice as to the propriety of surgical intervention for the detachment. In view of the probably tuberculous nature of the disease underlying the detachment, Bujadoux feels that a happy termination has been achieved.

<div align="right">L. L. Mayer.</div>

FAMILIAL MACULAR DEGENERATION OF UNKNOWN CAUSE. H. TILLÉ, Bull. Soc. d'opht. de Paris, July 1936, p. 548.

In two young girls, sisters, of a family of eight children, bilateral macular degeneration which had begun apparently at birth had resulted in loss of central vision. Tillé feels that this condition does not fit in with that described as due to syphilis by Morax in France, with that due to tuberculosis as shown by authorities of central Europe or with that caused by abiotrophy or focal infection as described by North American writers. Photographs of the macular regions of both patients are shown. All physical examinations showed no evidence of disease, and the antecedents in two previous generations had normal eyes. Treatment was of no avail. Tillé is unable to classify the lesion in these patients with any reported in the previous literature, which he reviews.

<div align="right">L. L. Mayer.</div>

TRANSITORY BLINDNESS FROM RETINAL ARTERIAL HYPERTENSION. D. CATTANEO, Ann. di ottal. e clin. ocul. 65: 81 (Feb.) 1937.

During an attack of mushroom poisoning a man of 36 noted sudden loss of vision. Vision was reduced to perception of light in each eye. The retinal vessels were somewhat small, and the disks were slightly pale. A few small hemorrhages were noted. The general blood pressure was 205 systolic and 115 diastolic, and the urine showed albumin and casts. The retinal blood pressure was estimated with Bailliart's dynamometer. In each eye the arterial pressure was 150 systolic and 105 diastolic. The venous pressure in the right eye was 15 mm. and in the left eye 20 mm. Intramuscular injections of acetylcholine had no effect on vision or on the retinal blood pressure. Three days after the onset of the visual symptoms 0.1 Gm. of acetycholine was given by retrobulbar injection. The next day vision had improved to 4/10 in the right eye and 9/10 in the left, and both the general and the retinal blood pressure had begun to diminish. A second retrobulbar injection was given. Five days after the onset of the symptoms vision was normal in each eye. The general blood pressure was 125 systolic and 80 diastolic. The retinal arterial pressure was 100 diastolic and 45 systolic for the right eye and 95 systolic and 40 diastolic for the left. The retinal venous pressure was 5 mm. in each eye. The renal condition cleared up more slowly but completely, while no further loss of vision was noted during the period of observation. Sudden hypertension with associated visual symptoms has not previously been reported as a result of mushroom poisoning but was evidently the condition in this case, since no signs of previous vascular disease were present. Cattaneo believes that a sudden vascular spasm as a result of the poisoning was responsible for the renal and vascular changes. A bibliography is included.

<div align="right">S. R. Gifford.</div>

RETINAL ANGIOSCOPY IN ARTERIAL HYPERTENSION AND RETINITIS OF PREGNANCY. C. E. LUQUE, Arch. de oftal. de Buenos Aires 11: 272 (May) 1936.

Luque stresses the importance of angioscopy in determining whether arterial hypertension appearing in pregnancy is of toxic (eclamptic) origin or is pseudo-eclamptic and the consequence of an aggravation of latent preexisting nephritis and, likewise, whether retinitis coming on in pregnancy is toxic or due to an exacerbation of latent nephritis. These points are easily determined by angioscopy, there being no lesion of the walls of the retinal vessels (best observed at the arteriovenous crossings) in the toxic varieties and more or less pronounced lesions in those varieties complicating chronic nephritis.

Six cases are reported: two of arterial hypertension in pregnancy in which the ophthalmoscopic findings confirmed the toxic nature of the condition as suspected by the obstetrician, two in which a suspected nephritic origin was confirmed and two in which the lesions of the fundus in the one patient and their absence in the other patient furnished the necessary data for a correct diagnosis, the other clinical symptoms pointing in another direction.

C. E. FINLAY.

CYSTIC DEGENERATION OF THE ORA SERRATA. GIOVANNI BRUNO, Arch. f. Augenh. 110: 183, 1936.

Bruno concludes that cystic degeneration of the retina is not a normal change due to senility, as a number of authors have asserted, but that it is a chronic edema which destroys the layers of the retina.

This edema is apparently caused by a change in the permeability of the capillaries at the anterior edge of the retina.

F. H. ADLER.

FERMENTS OF THE RETRORETINAL FLUID IN RETINAL DETACHMENT. H. WEVE and F. P. FISCHER, Arch. f. Augenh. 110: 198, 1936.

Weve and Fischer collected the retroretinal fluid from a series of patients with retinal detachment at the time of operation. Diathermy needles were used for making the punctures. All the fluid was examined for red blood cells and discarded if any were found. Although there was a possibility of contamination with lacrimal fluid, tests showed that lacrimal fluid itself never contained starch-splitting ferments. In a series of cases it was found that in a number amylase was present, which came from the retina. In another group of cases of detachment of the retina the retroretinal fluid contained no amylase. It was not possible to distinguish clinically between the two groups. Amylase was found in eight of twenty cases.

F. H. ADLER.

BLOOD PRESSURE IN THE CENTRAL RETINAL ARTERY. S. SUGANUMA, Klin. Monatsbl. f. Augenh. 97: 498 (Oct.) 1936.

This continuation of the first report, which appeared in a previous issue of the *Klinische Monatsblätter für Augenheilkunde* (96: 74 [Jan.], 1936; abstr., ARCH. OPHTH. 16: 880 [Oct.] 1936). In the present

article Suganuma reports on the blood pressure in the central retinal artery in divers forms of general hypertension and in so-called isolated cephalic hypertension. Tables with data on the results of urinalysis, the blood pressure and the appearance of the fundus are included, and an extensive bibliography is appended. The relation of the systolic pressure in the central retinal artery to the general blood pressure was 56: 100 in essential hypertension and 51: 100 in chronic nephritis and in secondary atrophy of the kidney.

Considerable fluctuations have been observed in essential hypertension. The relation of the pressure of the pulse to the maximal pressure, i. e., the coefficient of the blood pressure, in the retina averages 0.28 in essential hypertension and 0.24 in nephritides. The capillary blood pressure in the macular region of the retina shows an irregular increase in general hypertension, normal values occurring occasionally. The latter result is contradictory to Kylins' readings. Suganuma places under the heading "isolated cephalic hypertension" all the cases in which the general hypertension was normal while the pressure in the retinal arteries was raised. This hypertension was found in divers specific diseases, such as increase of the cerebral pressure, nephritis in pregnancy, epilepsy and retrobulbar neuritis. It occurred, furthermore, in certain stages of essential hypertension and of chronic nephritis. Therefore this hypertension is not rare, but it is readily overlooked, in the author's opinion. Variations of the caliber, reflexes and course of the retinal vessels are not dependent on the height of the blood pressure in the retinal artery. Arteriovenous compression (Gunn's or Salus' phenomenon) is generally due to an increase of the tonus in the wall of the artery, i. e., an increase of the retinal blood pressure. It is especially high in the presence of organic changes in the walls of the vessels. Its degree, however, is not dependent on the height of the local retinal blood pressure. A reliable diagnosis regarding the intra-ocular and intracranial circulation is possible, in the author's opinion, only on measurement of the blood pressure in the retinal vessels, aside from the customary general ophthalmoscopic examinations.   K. L. STOLL.

Trachoma

BACTERIOLOGICAL AND EXPERIMENTAL RESEARCHES ON THE AETIOLOGY OF TRACHOMA. A. CUÉNOD and R. NATAF, Brit. J. Ophth. 21: 309 (June) 1937.

The authors give the following summary:

"My first researches were made in 1907 in Tunis. They have established that trachoma is a contagious disease, caused by a virus the principal characters of which have been described above.

"Under the influence of researches on typhus and on the Rickettsias in various bacteriological centers, and especially at the Institut Pasteur de Tunis, bacteriological and experimental researches have been made showing that the trachomatous virus has a close relationship with an infra-organism of the Rickettsia family, the presence of which is morphologically demonstrable, as Busacca as well as ourselves has pointed out.

"The anal inoculation of the louse with trachomatous material causes the rapid increase of the organism. After many passages it is still able to reproduce lesions characteristic of trachoma on the conjunctiva of the Barbary ape (singe) and of man.

"Therefore it seems to be proved that trachoma is due to an infection of the conjunctiva with Rickettsias, these being the active principle of the trachomatous virus.

"We consider that we have proved both morphologically and experimentally that trachoma is a Rickettsiosis, and that the louse forms a reservoir, and is perhaps one of the vectors of trachomatous virus, probably the principal one."

W. Zentmayer.

Third Note on the Bacterial Agent of Trachoma. A. Cuénod and R. Nataf, Arch. d'opht. 53: 218 (March) 1936.

In this report Cuénod and Nataf consider the pathologico-anatomic features as shown by sections of trachomatous tissue which corroborate and clarify the clinical observation made by the use of smears. They have observed a powdery coccobacillary figure filling the spaces between the hypertrophied nodes of epithelioid cells which can hardly help being thought of as the multiplication of a parasitic agent of lower virulence which is of such a nature as to account for the pathologic hyperplasia of the nodes as well as the cytolysis of the peripheral cytoplasm of the epithelioid cells. Consideration of these cells suggests the idea of epitheliosis. Some support is gained from the latest work of Morax and especially the work of Thygeson.

S. B. Marlow.

The Pathology of Trachoma. R. P. Wilson, Bull. Ophth. Soc. Egypt 29: 1, 1936.

The characteristic infiltration in trachoma is composed of lymphocytes, mononuclear leukocytes, plasma cells and histiocytes. Polymorphonuclear leukocytes are present only in mixed infection. Since the expressible follicles of the second stage of trachoma are found chiefly in the retrotarsal folds, they cannot be the sequelae of strangulation of the meibomian ducts, as no meibomian ducts are found in this situation.

J. E. Lebensohn.

Tumors

A Case of Dermo-Epithelioma of Parinaud. G. Renard, R. Huguenin and P. Cassiau, Arch. d'opht. 53: 197 (March) 1936.

In 1884 Parinaud described a tumor "reddish yellow in color, semitranslucent, sometimes lobulated, situated outside the margin of the cornea, movable on the sclera, and occurring in young subjects," to which he gave the name dermo-epithelioma. The authors have collected fifty-two instances in which there was an essentially similar histologic picture but in which the tumor was described under varying designations. They report in detail the results of observation of a man 67 years old

who ten years before had had subconjunctival injections and other treatment for detachment of the retina. He had seen no physician for seven years prior to observation. A tumor was present in the conjunctiva near the caruncle, which after removal was revealed by histologic study to be a dermo-epithelioma closely resembling that described by Parinaud. As a result of the study of all the observations reported the following facts are noteworthy: 1. The tumor is almost always situated in an exposed portion of the conjunctiva. 2. In three cases it has been associated with a pterygium. 3. In four cases there has been evidence of trauma to the conjunctiva at the site of its occurrence. While the term dermo-epithelioma is not perfect, the authors believe it to be better than any other so far proposed. The diagnosis is simple; there is no conjunctival irritation or pain. Excision usually results in complete cure.

S. B. Marlow.

A Case of Lymphocytoma of the Orbit. M. de Treigny, Bull. Soc. d'opht. de Paris, June 1936, p. 406.

A patient aged 56 years had exophthalmos of the right eye of three years' duration. Surgical intervention had been instituted one year previously. The proptosis was direct, nonpulsating and nonreducible and gave the impression that a mass was behind the globe. Visual acuity was 0.7, and the fundus was entirely normal. During a year's observation the exophthalmos was slightly increased, and on palpation a firm lobule presented itself at the inferior external angle of the orbit. The conjunctival vessels were greatly dilated; the globe was only slightly movable, and the center of the cornea was ulcerated and stained. A roentgenogram showed a soft shadow of the entire orbit and the sphenoidal opening. Operation performed through an orbital incision at the external canthus for the purpose of removing a specimen for biopsy failed to alter the exophthalmos. Histologic examination revealed a homogeneous mass of lymphoid tissue with some large fibrous strands running through it. The cells were lymphocytes with a great number of mitotic figures and some large cells with much clear reticulum. Application of radium on the external portion of the orbit was instituted. Ten applications were given; after the second amelioration began, and there was complete reduction of the exophthalmos at the end of the course of treatments. Slight limitation of the movements of the globe persists. General examination gave no positive findings. The observation illustrates the relatively benign nature of these tumors and their extreme radiosensitivity.

L. L. Mayer.

Preoperative and Postoperative Ocular Symptoms of Tumors of Rathke's Pouch. F. Spinelli, Riv. oto-neuro-oftal. 13: 489 (Nov.-Dec.) 1936.

Spinelli describes eight cases of Rathke's cysts before and after surgical intervention. The purpose of this study was to establish whether the ocular findings could furnish enough data to enable one to localize the tumor. The majority of modern authors are of the

opinion that tumors of Rathke's pouch develop between the anterior and the posterior lobe of the hypophysis. They can therefore cause pressure on the hypophysis, on the optic chiasm and on the optic pathways. If they develop more extensively they may damage the third ventricle, the cavernous sinus, the cerebral peduncles or even the internal carotid artery.

In Spinelli's eight cases operation was performed by the frontotemporoparietal method, and Rathke's cysts were found varying in size from that of a small nut to that of a chicken's egg. In almost every case the cyst was adherent to one or both optic nerves, causing pressure on them. Increased intracranial pressure was noted in two cases, headaches in five cases, vomiting in three, vertigo in one and polyuria in one. Differences in vision in the two eyes were noted. In two cases vision was 10/10 in each eye. However, in the others there was a definite difference between the two eyes. In one case vision was 20/20 in one eye and in the other was reduced to perception of movements of the hand. The visual field varied. In seven cases it was affected bilaterally; in one case the one eye was normal, while in the other there was only residual vision in the inferonasal quadrant. Classic bitemporal hemianopia was found in only one case; in another case there were absolute temporal hemianopia in the right eye and relative temporal hemianopia for white and total achromatopsia associated with a relative central scotoma for white in the left eye. The fourth patient presented concentric contraction of the fields. The fundi showed either temporal or descending atrophy of the optic nerve. In one case there was papillary edema. The roentgen rays showed constantly lesions of the sella turcica, especially in the posterior portion. It is noteworthy that the age of these patients ranged from 6 to 29 years.

Spinelli concludes that while examination of the fundus, accurate determination of the visual fields and screen tests are of importance, one must also make an accurate study of the x-ray plates so as to localize the tumor and proceed to surgical treatment.

In every case, after removal of the cyst the atrophic change in the optic nerve was arrested.

<div align="right">G. Bonaccolto.</div>

Tumors

Prognosis of Sarcoma of the Uvea. H. Denecke, Klin. Monatsbl. f. Augenh. 97: 594 (Nov.) 1936.

Denecke refers to previously published observations on the prognosis of uveal sarcoma, with which his own observations coincide. These observations were made on thirty-six patients treated at the University Eye Clinic of Greifswald within the last twenty years. Their ages ranged between 40 and over 70 years, the decade of 51 to 60 years being chiefly represented. Sarcoma of the choroid predominated, being present in twenty-six patients; the ciliary body was afflicted in four and the iris in two. Sarcoma of the ciliary body showed relatively the largest number of metastases, namely three in four cases. Fourteen patients died from metastases, eleven of these within five years after enucleation, and one of them fourteen and one-half years after this operation.

Denecke considers the first five years after enucleation as the critical period, which should be considered in writing life insurance policies. The risk is small after the lapse of ten years.

Pigmented sarcoma showed no greater tendency toward metastases than the nonpigmented type; in nineteen of the thirty-six cases the tumor was pigmented. Exenteration of the orbit was performed in seven cases; in such cases the prognosis is not absolutely unfavorable, as one of these patients was still living after twelve and one-fourth years. In one patient perforation into the orbit was histologically noted after enucleation; he was living thirteen years afterward, although no exenteration of the orbit was performed. Local recurrences were not observed.

K. L. Stoll.

Visual Tracts and Fields

Angioscotomas. H. Viallefont and R. Lafon, Arch. Soc. d. sc. méd. et biol. de Montpellier 17: 122, 1936.

Using each other as subjects, the authors plotted the angioscotomas before and after inhaling amyl nitrate. No change was noted.

J. E. Lebensohn.

Visual Tracts and Fields

A Case of Double Hemianopia. J. Malbrán and E. de la Riega, Arch. de oftal. de Buenos Aires 11: 538 (Sept.) 1936.

The authors report a case of double hemianopia with preservation of the macular region and a quadrant below. The literature is extensively reviewed, and attention is called to a similar condition reported by Wilbrand and Sachs and classified under type XXI in the classic work of Wilbrand and Sänger.

C. E. Finlay.

Therapeutics

Utilization of Sodium Hyposulfite in Ocular Therapeutics. A. Dubois-Poulsen, Bull. Soc. d'opht. de Paris, July 1936, p. 560.

Sodium hyposulfite has been used for a long time in general therapeusis. In 1860 Polli experimented with its protective properties. The revival of its use was inaugurated by the work of Ravaut in 1920. In intolerance, anaphylaxis and allergic states it has been of much value as a desensitizer. Its use for eczema is well established. Dubois-Poulsen has found the drug efficacious in eczema of the lids, blepharitis, certain forms of conjunctivitis and certain forms of keratitis. He lists a group of patients who have benefited by this therapy. No untoward results or complication were observed. A review of the pharmacodynamics of sodium hyposulfite is given.

L. L. Mayer.

VALUE OF ATROPINE IN CASES OF TABETIC ATROPHY. J. FEJER, Arch. f. Augenh. **110**: 76, 1936.

No improvement in the visual acuity could be found in cases of tabetic atrophy of the optic nerve following retrobulbar injections of atropine, as advised by Zamkovsky and by Springovitsch (*Klin. Monatsbl. f. Augenh.* **90**: 343, 1933). In those patients who were followed over a long period it seemed to Fejer that the atropine had some beneficial effect in staying the disease process. Favorable results were observed in atrophy of the optic nerve following retrobulbar neuritis when this method of treatment was used. F. H. ADLER.

Society Transactions

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

Annual Congress, London, England, April 29 and 30 and May 1

Dr. Gordon Holmes, C.M.G., C.B.E., F.R.S., *President*

The Prognosis in Papilledema. Dr. Gordon Holmes.

Papilledema is now recognized to be essentially edema of the nerve head, in the early stages of which there is little structural damage or functional disturbance of the nerve elements, which later may be strangled and destroyed by overgrowth of interstitial tissue. In the latter stage, recovery of vision is not to be expected. The essential etiologic factor in papilledema is increased intracranial pressure. Relief of that pressure, if effected before the development of secondary changes in the disk, will lead to disappearance of the condition and so will remove the risk of blindness. In rare cases the papilledema subsides without surgical intervention or other specific treatment, and vision remains unaffected.

In most cases of papilledema, relief of intracranial pressure becomes necessary in order to save sight. The more rapidly the congestion and swelling of the optic disk develop, the more intense becomes the papilledema and the greater the danger to sight if relief is not undertaken. Swelling of 4 or 5 D. is a warning against undue delay. Equally serious are great engorgement of retinal veins and early and extensive hemorrhages on the surface of the swollen disk. An even more important danger is narrowing of the arteries in the swollen disk and its vicinity. Frequent examination should be made in order to evaluate any changes occurring in the fundus and to detect the earliest signs of atrophy of the optic nerve. A further serious sign is the transient loss of sight, which may occur after sudden change of posture. If the tumor or other cause of the increased pressure cannot be removed, decompression craniectomy usually causes subsidence of the congestion in from two to four weeks, and if secondary changes have not appeared by then there is no further risk of impairment of vision.

Retinal Circulation: Changes in Metabolic Disease. Mr. A. E. MacDonald, Toronto, Canada.

From the material presented the speaker drew the following conclusions: 1. Papilledema which is indistinguishable from that in cases of tumor of the brain occurs in some cases of metabolic disease. 2. In the latter the eye is subject to injury by toxins in equal degree as other parts, but the outstanding retinal changes cannot be explained by toxins, arteriosclerosis or inflammation. 3. Obstruction of the ocular drainage develops with an explosive, a slow or an intermediate time factor. 4. Venous distention at the disk occurs late in systole and disappears when the intracranial pressure is raised to about 250 mm.

of water or 18 mm. of mercury. 5. Straining, lifting, coughing and holding the breath produce elevation of the intracranial pressure sufficient to cause obstruction of the central vein.

CATHOLYSIS IN THE TREATMENT OF RETINAL DETACHMENT. MR. R. FOSTER MOORE.

The object of catholysis is to produce coagulation in the choroid as the result of the liberation of sodium ions at the negative terminal inserted through the sclerotic. At the same time hydroxyl ions are set free and can be readily seen with the ophthalmoscope during the operation. The platinum iridium needles which I use are either straight or bent at a right angle. The length of the active part of the needle varies from 1 mm. upward. The needles can easily be passed through the muscles into the globe, and when used thus must be from 4 to 5 mm. in length. The optimum strength of current is 5 milliamperes, applied for six seconds. An almost unlimited number of punctures can be made, even though the eye may be somewhat soft. Bubbles of gas are seen at the site of the puncture, and from the punctures there is always some escape of fluid. Frequently no additional exit is required for the outflow of subretinal fluid.

Of 31 patients treated by this method, 14 were myopic. In 12 the retina was replaced, and was in position when the patient was last seen. Two patients were discharged slightly improved, 4 much improved and 9 not improved. The longest posttreatment period has not been over twelve months and in many of the cases has been only a few weeks. The advantages of the method are that the apparatus required is light, portable and easily used, and the effects of the procedure are sharply localized, with less damage to intra-ocular tissues than results from diathermy. It is unnecessary to divide the muscles unless exposure of the sclerotic far back is required. The crucial test for success is not in how many cases the retina goes back but in what proportion the replacement is permanent.

CRITICISM AND COMMENT ON NEWER METHODS OF TREATING DETACHMENT OF THE RETINA. MR. J. COLE MARSHALL.

In catholysis the whole length of the needle is active, whereas in diathermy the only active part of the needle is its base, and often the point becomes cool. In catholysis, keratitis and retinitis are formed beyond the region where the bubbles have been liberated, so that when the retina goes back the retinitis in that region may cause changes in the choroid. Mr. Foster Moore retains the needle in the eye longer and uses a slightly stronger current than do most continental operators. Certainly catholysis is a good localizing agent, and it is also a good adjunct to diathermy scarring. In cases of detached retina I have found catholysis very beneficial when used in conjunction with diathermy, and for the latter the use of very fine needles represents a distinct advance. The transillumination method described by Weve I regard as very important, as is also the continuous use of saline solution during the operation.

Exophthalmic Ophthalmoplegia. Dr. W. Russell Brain.

The syndrome of exophthalmos and ophthalmoplegia may occur either unilaterally or bilaterally. When arising spontaneously it is associated with general symptoms of thyrotoxicosis. But it may follow thyroidectomy for hyperthyroidism, in which case general symptoms of thyrotoxicosis are often absent, and the basal metabolic rate is normal or even subnormal.

The separation of this syndrome from exophthalmic goiter depends on a number of features, the most important being that it may occur postoperatively, not only in the absence of thyrotoxicosis but in the presence of actual hypothyroidism. It also differs from exophthalmic goiter in its age incidence and its sex incidence, in the usually slight degree of the thyrotoxic symptoms, when these are present, in the lack of response to thyroidectomy and in the somewhat atypical histologic picture presented by the thyroid. As to the period of life at which cases are observed, exophthalmic ophthalmoplegia is a disorder of middle age. The mode of onset is usually subacute: One eye becomes increasingly prominent over a period of from three to four months, ophthalmoplegia and double vision developing concurrently. Generally the other eye lags behind in the matter of proptosis and may not exhibit ophthalmoplegia. Both exophthalmos and ophthalmoplegia may develop simultaneously in each eye in three or four months. Exophthalmos was present in all my cases; it was unilateral in 5 and bilateral in 24. In the latter cases the degree of exophthalmos of the two eyes was equal in 8 instances and unequal in 16. Asymmetry of the proptosis was present in 21 of 29 cases. Ophthalmoplegia was unilateral in 12 of the cases and bilateral in 17. The ophthalmoplegia was paresis or paralysis affecting not movements of individual extra-ocular muscles but movements of the eye in a particular plane. Among the cases in which the ophthalmoplegia was unilateral, elevation was the movement most often affected; in the cases in which it was bilateral, abduction was affected in 31, elevation in 23, depression in 18 and adduction in 17. In 6 patients all movements of each eye were affected.

Widening of the palpebral fissures associated with retraction of the upper lid was the usual finding in both cases of unilateral and those of bilateral ophthalmoplegia, but proptosis was present on each side in 5 cases and on one side in 3 cases of bilateral ophthalmoplegia.

With regard to the pathologic changes of the ocular muscles and the thyroid, these consisted of marked edema, with foci of lymphocytic infiltration and, in later stages, fibrosis. In one portion of the levator palpebrae superioris muscle, which was removed at operation, Professor Turnbull noted general edema and great enlargement of the muscle fibers. In 4 of 5 cases, sections of the thyroid showed that the gland was atypical, and in the other case the gland was in the same condition as in cases of exophthalmic goiter.

In my experience, the usual treatment for exophthalmic goiter has been disappointing when applied for exophthalmic ophthalmoplegia. Thyroidectomy was done in 4 cases. In 1 of these cases there were marked general improvement, and considerable improvement in the condition of the eyes, though some ophthalmoplegia remained. After

orbital decompression immediate recession of the eye occurred, but the ophthalmoplegia remained unchanged in 1 case and was only moderately benefited in the other 2. I hope the day will come when the condition can be dealt with physiologically.

PROF. F. R. FRASER: I have been able to test the effect of prostigmin on 2 patients with exophthalmic ophthalmoplegia. During the last twenty-five years there has been a great advance in the knowledge concerning the transmission of nerve impulses to muscles and especially as to the importance of acetylcholine. This substance, it has been shown, is essential for the transmission of nerve impulses across the ganglions of the sympathetic system, for the transmission of impulses from the vagus nerve to the effector organs of the vagus system and also for the transmission of nerve impulses from nerve endings to voluntary muscles. But the substance is rapidly destroyed in the body; otherwise, the muscle would remain in a state of tetanic contraction. It is destroyed in the presence of choline esterase.

It has frequently been noted that there are similarities between toxic goiter and myasthenia gravis. Many persons who suffer from the former disease complain of severe muscular weakness and excessive fatigability; the similarity is most marked in patients with goiter in whom exophthalmic ophthalmoplegia develops. In both cases of toxic goiter and those of myasthenia gravis, lesions of the thymus gland have been recorded, and on histologic examination the muscles, in both cases of myasthenia gravis and those of ophthalmoplegia, show collections of lymphocytes. Dr. Mary Walker has recently shown that a synthetic analog of physostigmine known as prostigmin restores voluntary muscle in cases of myasthenia gravis. The effect is dramatic, as it is evident a few minutes after this drug has been subcutaneously injected. In a few hours the effect passes off. This action is due to the inhibition by prostigmin of the esterase. No abnormality in the choline esterase of the serum was present in myasthenia gravis, and a disturbance of the normal balance between the production of acetylcholine and the activity of the esterase at the neuromuscular junction is supposed to exist.

The first patient with exophthalmic ophthalmoplegia on whom prostigmin was tried was a woman aged 41 who at the age of 26 had begun to show signs of toxic goiter; those evidences were tremors, palpitation and marked sweating. A year later she showed exophthalmos, which was more marked on the right than on the left, diplopia and pronounced ptosis of the left upper eyelid. The ophthalmoplegia developed rapidly and interfered with her work, which was teaching school. She had at this stage drooping of the eyelid, which became more marked toward the end of the day, and at the end of the school term the diplopia was much more severe. In 1925 partial thyroidectomy was carried out, and after this the ophthalmoplegia became much better, and the ptosis on the left was so much improved that she was able to resume her teaching. The tachycardia and tremulousness, however, never disappeared. She was, therefore, treated with prostigmin, 1 mg. of atropine having first been used as a preliminary medicament. Twenty-three minutes after administration of the atropine, 2.5 mg. of prostigmin was given, a large dose, the possible upsetting effects of which were

counteracted by the atropine. Four minutes after the prostigmin was given there was improvement in the upward vertical movement of the right eye; in a few minutes the axes of the eyeballs became parallel when the patient looked straight forward; there was twitching of the orbicularis muscle, and diplopia was present only when she looked above the horizontal level. Seventeen minutes after the injection, diplopia was to be found only on extreme upward elevation of the eyes. At that stage the patient's condition began to retrogress a little, as shown by the fact that diplopia could be elicited more easily, and after half an hour the axes were again deviated. She said that during the rest of the evening her eyes were stronger. A week later a similar dramatic result was brought about in the same manner.

The second patient was also a woman aged 41, who had had toxic goiter when she was 32, the onset of which was marked by lack of energy and a feeling of weariness. A year later, exophthalmos was noticed, but there was absence of severe toxicity. Then diplopia appeared. Thyroidectomy was done in 1930, and since then the ophthalmoplegia has remained stationary. During the last few years the patient has been treated with thyroid because of her feeling of lassitude and general depression, and in the last year the exophthalmos has increased to a greater degree than at any time before. Prostigmin was used, but in this patient it had no effect.

The difference as to the result of treatment with prostigmin in these two cases suggests a different condition in them, or it means that the disease process in the second patient had progressed to such an extent that it could not respond to the drug.

A Case of Exophthalmic Ophthalmoplegia. Mr. H. B. Stallard.

A man aged 31 complained that on looking upward and on opening the eyes after they had been closed two or three minutes he had diplopia. His condition was becoming progressively worse. He had a tired feeling in his eyes and general lassitude; he was also nervous and easily worried. There was evident bilateral exophthalmos, which was more marked on the left. The thyroid was slightly enlarged, soft and of uniform consistence. The pulse rate varied from 64 to 90, the reflexes were brisk, and the basal metabolic rate was — 40 per cent. No abnormality was found in the blood count or from a roentgenogram of the skull. The Wassermann reaction was negative. The pharyngeal lymphoid tissue was reported to be inflamed. Injection of 600 organon units of the thyrotropic hormone of the anterior lobe of the pituitary was given on three successive days, to permit study of the effect of that substance on the activity of the thyroid and the basal metabolic rate. The latter rose from 3 to 21 per cent within twenty-four hours after the first injection was given; a further rise to 30 per cent took place after the second and third injections, and in three days the rate rose to 41 per cent. During the twelve hours after the first injection the temperature rose, and there was a corresponding increase in the pulse and respiration rates. Frontal headaches occurred ten hours after the first injection and eleven hours after the second and third. Then the patient slept; the next morning the headache was only slight, and it did

not occur again. Three weeks later a corneal ulcer developed in the left eye, and in spite of tarsorrhaphy, phenolization and paracentesis this eye lost its sight.

DISCUSSION

DR. GORDON HOLMES: In a certain proportion of the cases of proptosis of the eyes which I have observed there have been few other symptoms of thyrotoxicosis, and, in particular, there was no increase in the pulse rate. It is possible that there are three types of this condition. Professor Fraser's results in 2 cases indicate two types, and I have seen instances of a rarer form, one characterized by definite weakness of some of the ocular muscles, with rapidly developing exophthalmos. In reports of cases of thyrotoxicosis some subluxation of the globe has been described. I think the transient palsies are caused by sudden stretching of the muscles, due to mechanical causes, in the orbit itself. As to the pathologic basis, Sir William Gowers, many years ago, mentioned the possibility of primary degeneration of the cells occurring in these cases. Vigor demonstrated leukocytic infiltrations in myasthenia gravis, but Mr. Stallard has now reported that in his case the muscle fibers did not show pathologic change. The question whether there is in this condition a change in the nerve supplies of the muscles has not been answered. In exophthalmic ophthalmoplegia there is some variability in the symptoms; several of my patients have said that their ocular movements were fuller in the morning than at the end of a tiring day. This exhaustibility of ocular movements brings this condition more into line with myasthenia gravis, a point which Professor Fraser has mentioned.

DR. RUSSELL BRAIN: In 1 case serial sections were made of the brain stem, and no abnormality was found in the nervous system. Some ophthalmologists advocate bandaging the eye, but I always found that this made the condition worse.

PROF. F. R. FRASER: Prostigmin reaches its full effect in half an hour after injection, and the effect remains obvious for four hours. When the method of injection is used, the drug has to be given every four or five hours. Less than 2.5 mg. is not of much use in myasthenia gravis. A much smoother action is obtained when the drug is taken by mouth, but with this method of administration large doses must be taken.

SPONTANEOUS CURE OF RETINAL GLIOMA. MR. MONTAGUE L. HINE.

Paintings were shown of three eyes in which nature had evidently cured early gliomatous growths. In the last edition of his textbook Sir John Parsons gave the prognosis of glioma of the retina as being wholly poor, the patient invariably dying of the condition if untreated. In 1933 Griffith recorded particulars of a "glioma family," and a fuller history was given in his report. The father, aged 42, had his left eye excised in infancy because of retinal glioma, and on recent examination his other eye showed on the nasal side of the fundus an atrophic area of the retina and choroid on which was superimposed a shrunken, slightly raised cystic mass of retina, which was presumably, if the history is borne in mind, a regressed and cured patch of old glioma. The

eldest child died at 6 months of age; the second child, now 19 years old, was found to have cystic masses in each retina, that in the right eye being typical of previously described spontaneously cured retinal glioma. The third child died of retinal glioma, which was bilateral, at 21 months of age; the fourth had one eye excised for the same condition when 3 months old, and the fifth died of bilateral glioma at 4 years of age.

DISCUSSION

Sir John Parsons: I consider that my general statement in my textbook for students on this matter is correct. I am very doubtful about recorded cases of spontaneous cure of this condition, and, in any case, it would be wrong and dangerous to lead students to expect nature spontaneously to cure this serious condition. Every case of so-called cure should be backed up by report of histologic examination by experts.

Mr. R. Foster Moore: I have observed a similar case of spontaneous cure. I think there can be no reasonable doubt about these instances of cure which have been reported.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

May 17, 1937

John H. Dunnington, M.D., *Chairman*

LeGrand H. Hardy, M.D., *Secretary*

Chancre of the Upper Eyelid in an Infant Two Months of Age. Dr. Alfred Appelbaum.

An apparently normal infant of 8 weeks showed on the right upper lid a sharply defined oval ulcer, with its longest diameter in the horizontal direction, 3 mm. high, mounted on a painless induration. The surface was level, dry, crustlike, and 0.5 mm. below the level of the border of the slightly edematous encircling skin. The corresponding preauricular glands were enlarged. The initial dark field examination gave negative results, but three days later, examination disclosed Spirochaeta pallida. The source of infection was late secondary syphilis, which appeared in the mother one month after delivery. She had had antisyphilitic treatment four months before pregnancy commenced, discontinuing the therapy shortly thereafter. The Wassermann reaction of the blood was negative just before and after midterm and at delivery.

Synchysis Scintillans in the Anterior Chamber. Dr. Girolamo Bonaccolto.

Synchysis scintillans in the anterior chamber was seen in a woman 64 years of age. Examination showed the right eye to be congested and hypertonic. Under the slit lamp myriads of spherical bodies of various dimensions were seen, from which many colored lights were reflected.

Other writers have reported these bodies as polygonal crystals. Seen with the naked eye they appear as gold flakes or crystals in the aqueous or the vitreous, but under the slit lamp they have a spherical appearance.

DISCUSSION

DR. WENDELL L. HUGHES: This reminds me of a case that my associates and I observed at the New York Eye and Ear Infirmary six months ago, in which the anterior chamber was entirely filled with crystals and secondary glaucoma developed later. We did a paracentesis, collecting the fluids that were withdrawn, and under the microscope these were shown to be cholesterol crystals. The glaucoma was controlled by removal of the material from the anterior chamber. This was apparently degenerated vitreous.

PAGET'S DISEASE OF THE SKULL. DR. W. GUERNSEY FREY.

A 49 year old man with Paget's disease of the skull (osteitis deformans) presented the following ophthalmologic findings: alternating squint, papillitis in a stage of beginning atrophy, contracted fields and enlarged blindspots. There was no evidence of changes at the orbital apexes on roentgen examination.

DISCUSSION

DR. MARTIN COHEN: I should like to ask whether the ocular changes are due to Paget's disease or whether the ocular manifestations might not be due to some vascular disturbance having no bearing on Paget's disease. In a number of cases of Paget's disease the fundi have been absolutely normal, and I reported a case in a man 70 years of age who had lesions of the fundus similar to those shown by Dr. Frey's patient this evening. Autopsy in that case showed the characteristic bony changes in the skull. The vascular changes in the eyes were attributed to marked capillary sclerosis of all the choroidal vessels, and this condition might explain the capillary hemorrhages which occurred in the fundus of one eye in my patient.

DR. JOHN H. DUNNINGTON: I have followed a woman with Paget's disease for many years. She has vision of 4/200 in one eye; the other eye has no perception of light. The changes in the fundus are not typical. There is some atrophy of the optic nerve, but it seems to me that the loss of vision is out of proportion to the atrophy of the optic nerve. A younger woman, who has very advanced Paget's disease, has retained vision of approximately 20/40 in each eye. She has peripheral contracture of her fields of 10 degrees and a certain amount of atrophy of the optic nerve. There are no vascular changes in the fundus of the younger patient, but there is marked sclerosis of the choroidal vessels in the older woman.

DR. W. GUERNSEY FREY: We should all like to know what causes the ophthalmologic symptoms in Paget's disease. I have talked to a number of neurologists, and they are largely of the opinion that the neurologic symptoms are due to pressure on the nerves. I am hardly convinced of that myself after what I have read in the literature. It appears to me, since it is not known what the cause of the

condition is in the first place and since the bony lesions seem to be trophic disturbances, that the same underlying condition may be responsible for the ocular signs.

Laurence-Moon-Biedel Syndrome.　Dr. H. Templeton Smith.

It is of interest to ophthalmologists that although only one of the five cardinal manifestations of the Laurence-Moon-Biedel syndrome is ocular, the first cases to be recognized and reported were observed by ophthalmologists.

Since Laurence and Moon first reported this syndrome in 1866, only about 80 cases have been reported. The characteristic findings are: (1) polydactylism; (2) obesity, which comes on after a few years of age, with the general appearance of pituitary imbalance; (3) genital abnormality of varying degrees; (4) a condition ranging from mental deficiency to imbecility; (5) atypical retinitis pigmentosa, and (6) frequently a familial tendency.

The etiologic factors have been lightly touched on by the observers. The first and most stressed factor is the hypophysis. In the series, the results of the basal metabolic test in 17 cases were reported. In 2 cases the basal metabolic rate averaged $+ 25$; in 4 it was normal; in 4 it was low, and in 7 it averaged $- 25$. A survey of the literature reveals only one autopsy, with no abnormal findings except those grossly observed in the living patient.

The case of a Puerto Rican boy 14 years of age, the third child of a normal mother, was reported. Two brothers were normal. The patient had enjoyed normal health and vision up to 5 years of age. The first sign of any abnormality was failure of vision, followed by general sluggishness and slowing of all reactions.

Examination showed a supernumerary maldeveloped finger on each hand, an unhealthy pallor, small hands and feet with no muscular strength, and obesity. The Wassermann and Kline reactions of the blood were negative, and the blood chemistry was normal. The hemoglobin content was 63 per cent, the red cell count 4,700,000 and the white cell count 8,700. The basal metabolic rate was $- 18$. A roentgenogram showed the sella turcica to be moderate in size, without erosions, and the clinoid processes did not encroach on the pituitary space. Owing to lack of intelligent cooperation of the patient, the amount of vision and the fields could not be determined. In attempting to cross the room with various obstacles in his path the patient would bump into each one. External examination of the eyes gave negative results; the pupils were equal and reacted to light. The state of accommodation was not determined. The media were clear, and the optic nerves were oval and had a slight waxy pallor. The lumens of the vessels were contracted and slightly irregular. Scattered throughout both retinas were areas of atypical patches of retinitis pigmentosa, with some degeneration of the retina.

DISCUSSION

Dr. John M. McKinney: I saw this patient with Dr. Smith at St. Luke's Hospital. The parents of such patients as a rule do not pay much attention to the polydactylism; the first thing that attracts

their attention is the poor vision, so the ophthalmologist is often the first physician to see these patients. That was so in the first cases of this syndrome to be described in 1866 by Laurence and Moon, the British ophthalmologists. I was interested in the fact that the famous English physician Jonathan Hutchinson also examined these patients. The original report was buried in the literature until 1922; it was dug out by Biedl, the eminent Czechoslovakian endocrinologist, and that is why his name is added to the syndrome. There has been a great deal of talk about the treatment. In some cases the patient has responded to thyroid and pituitary therapy, but such treatment is apparently empirically used, for, as far as can be made out, the condition is not glandular in etiology but is probably a congenital defect on the basis of a defect in the germ plasm. This is borne out by the fact that in the majority of cases the disease is familial; that is, one or more members of the same generation are affected; but the syndrome is not hereditary.

In the interest of differential diagnosis, the patients are nearly always children when first seen, and if there is retinitis pigmentosa associated with striking obesity of the hypopituitary type, small genitalia, polydactylism, mental deficiency and familial incidence, the case falls into this diagnostic group.

Dr. Harry V. Judge: Appreciating the rarity of this syndrome, I desire to mention a case observed about three years ago. A child aged 4, the first-born of Jewish parents who were second cousins, was brought to me on account of convergent strabismus of the left eye. The child presented in the main the symptoms outlined by Dr. Smith, namely, marked mental deficiency, polydactylism, syndactylism, obesity, undescended testicles and a rudimentary penis. There were an extra digit on the right hand and an extra digit on each foot. The mental state precluded determination of the visual acuity. There was a fine lateral nystagmus with an occasional vertical component. On refraction the retinoscopic test showed need of a — 7.50 D. sphere for the right eye and of a — 9.0 D. sphere for the left. The fundus did not present the usual atypical retinitis pigmentosa as seen in the patient exhibited tonight, but the retina had the appearance of one lacking normal structure and showing tessillation with deeply pigmented islands between the vessels of the choroid—a picture somewhat similar to that in the case reported by Savin (*Brit. J. Ophth.* **19**: 597 [Nov.] 1935) but without the pigmentary changes noted in that case. It is possible that pigmentary changes may take place at a later date. The disk was dirty yellow and showed a sharp outline and contracted vessels. It is possible that central pigmentary changes were present, but, owing to the nystagmus and the poor cooperation of the patient, it is impossible to say with certainty. Two years ago an attempt was made by surgical intervention to bring the testicles within the scrotum, but the operation was unsuccessful. Prior to this, an extract of urine of pregnant women had been administered but resulted only in development of pubic hair.

The pictures show the characteristic stigmas. The members may have difficulty in observing the bilateral inguinal scars.

At the present time at the Albany Medical College some work is being done by Dr. James Hamilton, the endocrinologist, with an androgen known as testosterone propionate (used by Ruzicka, of Basel,

Switzerland) in cases of cryptorchidism. His results in studies on animals have been startling, and in the case of one human being he has reported a decided improvement in this condition.

My associates and I are anxious to have this drug tried in this case of the Laurence-Moon-Biedl syndrome to ascertain the effects on the genitalia, but we believe the action of the drug will be somewhat inhibited on account of the previous surgical intervention. The second boy in this family also shows cryptorchidism, but a cursory examination of his fundus showed none of the signs present in his brother's. The cryptorchidism may be an expression of familial hypopituitarism.

RELATION OF AMETROPIA TO OCULOMOTOR ANOMALIES. DR. JAMES W. WHITE.

The many exceptions to the general rules for the correction of ametropia, due to anomalies of the ocular muscles and accommodation, have not been stated. The reasons for the need of glasses were stated, and it was explained why ametropia may be better uncorrected, overcorrected or undercorrected, depending on the result of the tests and the presence or absence of symptoms.

DISCUSSION

DR. JOSEPH I. PASCAL: Dr. White advised correcting myopia fully when it is associated with exophoria. This is the old and standard teaching, and such correction is unquestionably feasible in a great many cases. It is predicated on the theory that the myopia is primary and the exophoria is secondary. But there is another side to this problem in cases in which the relationship seems reversed, that is, in which the exophoria is primary and the myopia secondary. Correcting the myopia fully in these cases without first treating the basic cause, i. e., the exophoria, would probably not give the best results. In early childhood, especially, it seems that such exophoria brings about functional overactivity of the associated accommodation, producing at first functional myopia, which in time becomes real myopia. In such cases correcting the myopia fully seems to work contrary to the principles suggested by the etiology and may lead to a further increase of myopia, whereas relaxing the overconvergence by correcting the exophoria with a prism base in or by some dissociating exercises seems to work more in harmony with the principles suggested by the etiology. It is also a fact that correcting the myopia fully generally does not lessen the amount of associated exophoria, which would also speak against the myopia being primary. Cases of primary exophoria with secondary myopia do occur, perhaps more often than is believed, and I should like to ask Dr. White what he thinks of these cases, drawing on his wide experience.

DR. JOHN H. DUNNINGTON: I am thoroughly in accord with everything Dr. White has said, particularly the fact that there are many myopes who do not care to wear glasses. I am a myope and prefer to wear my glasses and see, but there are many people who do not. I cannot understand why the ophthalmologist should insist on the use of glasses when the patient does not wish to wear them, unless the condition has some pathologic bearing on a muscular anomaly. It has been my experience that moderate myopia does not increase because

of failure to wear the full correction, nor does exophoria become worse under these conditions.

Dr. James W. White: In regard to theories of exophoria and myopia, I shall refer to my first statement or two, that rules and theories are all right if they work. If one finds an instance in which exophoria seems to be primary and myopia secondary, all right. If one finds an instance in which myopia is primary and exophoria secondary, all right, too; one should get proof of it. But I emphasize again one of my first statements: One should examine the patient before giving the cycloplegic, and then after, and as many times after that as is thought necessary. One should examine the patient under different conditions and see how the amount of deviation varies. Granted that one will find patients with primary exophoria who have myopia, I also know that one will find a good many patients with myopia in whom exophoria develops.

Operative Injuries of the Eye. Dr. Percy Fridenberg.

Operative injuries of the eye occur not only in ophthalmic surgical procedures, but in operations on the accessory sinuses, intranasal instrumentation and obstetric procedures. All forms of traumatism, from corneal abrasion to laceration of the optic nerve, and material and personal hazards in ophthalmic operations were noted.

DISCUSSION

Dr. Martin Cohen: The one point which struck me during the presentation of the paper was the nonuse of the speculum. It seems to me that operative procedures on the muscles, or extra-ocular procedures, require a speculum. I saw in Paris a procedure for extraction of cataract by Frouseau in which he separated the lids with the thumb and forefinger, introduced the cataract knife in the usual way, did a capsulotomy and then gently expressed the lens. He used no speculum and applied his thumb on the cornea to express the lens. I have seen him perform that procedure frequently, without any ill effects on the globe, which shows the importance of avoiding the use of the speculum in operations on the globe as much as possible.

Dr. Ernst Waldstein: Czermak in his later period used a speculum, and the assistant held the lids with his fingers only in exceptional cases. Elschnig utilized his assistants' fingers for holding the lids very sparingly; his first intracapsular extractions after Smith's method were performed with this precautionary measure. For my part, I have always doubted its advantage compared with the use of the speculum, especially when one keeps the latter unlocked, as Elschnig always urged. In order to get a good hold of the lids the fingers have to grasp them close to the edge, and then it is often almost next to impossible to avoid any undue pressure, the very thing one tries to prevent.

Dr. Percy Fridenberg: The data as to the use of the speculum were taken from the classic treatises on the operations on the globe. They do not take into consideration the operation for squint, because in operations on the extra-ocular muscles there is no special danger, even if the patient should squeeze a little, as the globe has not been opened.

There are one or two other points that I should like to add. In regard to the operation for squint and tenotomies, there are some interesting observations by Hermann Knapp in his treatise in Norris and Oliver's "Textbook of Ophthalmology." He stated that penetration of the globe in the course of tenotomy comes from using scissors with needle points, which are unnecessary, and working from underneath, obliquely. Knapp stated that this subject came up in a meeting of the American Ophthalmological Society. H. Derby said he had had several patients in whom this had occurred; in his practice Knapp said he had had 1 or 2, and after that a number of the older members admitted to having observed several cases in their own practices in which this had occurred.

GERMAN OPHTHALMOLOGICAL SOCIETY

Fifty-First Annual Meeting, Heidelberg, July 6-8, 1936

Translated by Percy Fridenberg, M.D., New York

Docent Dr. M. Bücklers, Tübingen, *Reporter*

SECOND SCIENTIFIC SESSION

Monday, July 6, 1936, 3 p. m.

Dr. W. Meisner, Cologne, *Chairman*

IV. Rôle of the Ophthalmologist in the Prevention of Hereditary Diseases. Dr. W. Clausen, Halle.

(Continued from page 332)

Patients with cataract associated with myotonic dystrophy should, without exception, be sterilized. The procedure appears fully justified in myopia of extremely high degree, so-called excessive myopia, in which severe alterations are noted as a result of traction in the region of the posterior pole at a comparatively early stage, especially if these pathologic changes allow one to foresee an early or precocious visual invalidism and if, in addition to the myopia, there is a certain hereditary tendency to retinal detachment. In closing, the reader of the paper discussed the practical results of the sterilization laws and stated that he saw promise of far reaching elimination of hereditary ocular disease by the consistent and appropriate carrying out of a campaign of eugenic preventive hygiene.

DISCUSSION OF REPORT IV

Hereditary Opacities of the Lens. Dr. M. Bücklers, Tübingen.

Inheritance of lenticular changes has been known for a long time. This applies not only to the juvenile forms but to the senile as well, as has been demonstrated lately by the results of Vogt's studies of identical twins. The reader was able to follow a number of patients with senile cataract through three generations and got the impression that the same

form is perpetuated in a given family. Of 5,047 patients operated on for senile cataract in his service, 17.6 per cent stated that this condition had occurred in their family. Social and heredobiologic importance attaches first of all to those lenticular changes which are congenital or interfere seriously with vision at a very youthful age. The large group of zonular cataracts belongs in this category. Jess was quite right in calling attention to the fact that even the most experienced ophthalmologist meets, again and again, to his surprise, with forms of zonular cataract previously unknown to him. The extent of the variations is shown in a small selected series of observations from the reader's services in Berlin and Tübingen.

Familial distribution has been, so far, determined only for the form which may have some relation to coralliform cataract. Whether one is dealing in a given case of zonular cataract in practice with a variation in manifestation of an already familiar basic type or whether hereditary factors are concerned and what these factors may be are questions which at the present time can be answered in each individual case only by expert and intensive investigation of the family histories and genetic research. This study meets with great difficulties, as the cataract is generally extracted without its appearance having been noted or even sketched for record. Anamnestic data are practically useless for this purpose. The genealogies which we have collected generally concern families in which there was numerical heaping up of cases. If we wish to avoid the dangers of such a "selection on the basis of interestingness" (Just) we shall have to carry out a comprehensive heredobiologic census of the population. For this we need the cooperation of each and every ophthalmologist. Only then shall we have light thrown on the occurrence of apparently isolated cases. The fact that zonular cataract has been produced experimentally in animals in various ways, such as by toxic agents, massage and radiant energy, has given the impression that this disturbance may also be due to exogenous factors.

We know that Horner accepted rachitis, and Peters and his disciples, tetany, as the cause. Considering the enormous frequency of these two diseases (80 per cent) in our regions, it was always probable that a chance coincidence would be mistaken for a causal relation. But, as far as I know, here the pragmatic test, viz., examining children with a previous history of severe rachitis or of tetany for evidence of the presence of zonular cataract, has never been applied to this problem. At my suggestion, Dr. Gscheidel has for this reason started a survey of children formerly treated in the pediatric clinic. So far, only 40 patients have been examined, but it is interesting to note that of these not 1 showed the slightest trace of zonular cataract. As a general proposition the reader was inclined to agree with Waardenburg that, especially in regard to hereditary lenticular opacities, we cannot individualize and differentiate sufficiently. It is only by individualization and differentiation that with industrious and intensive research we shall finally get a clear conception of the nature of the multiple relations concerned in pathologic changes of the lens. The fact that we are able at all to recognize fine differentiating characteristics of cataracts today we owe primarily to slit lamp biomicroscopy and its founder, Alfred Vogt.

STERILIZATION OF SUBJECTS WITH CONGENITAL CATARACT. DR. JESS, Leipzig.

In their comments on this subject, Gütt, Rüdin and Ruttke said: "Preventive eugenics must be considered primarily in all families in which we note early and widespread occurrence of a cataract type. . . . Furthermore, those subjects should be sterilized who show, in addition to congenital cataract, other indications of physical or mental inferiority, such as cerebral disturbances and similar conditions. In these cases of congenital cataract accompanied with other 'complications' we know that operative procedures offer a very unsatisfactory prognosis as to ultimate vision . . . At the very least, all subjects with so-called complicated congenital cataract should be rendered incapable of reproducing." Fleischer was then cited to the effect that a good visual result is generally not to be arrived at even with a correctly performed and uneventful operation for congenital cataract. This author then went on to say: "From this welcome point of view, in accordance with which the decisions as to sterilization must be made," etc. In accord with this dictum, as experience in a large number of cases has demonstrated to the reader, many practitioners have been convinced that the law requires the patient with any form of cataract recognized as definitely congenital to be sterilized without any further consideration. Certainly, every case of cataract recognized as congenital and hereditary must be reported to the district health officer. However, the decision as to sterilization concerns the judicial body (*Erbgesundheitsgericht*), which in each case bases its decision on the careful and detailed reports of the expert medical examiners. It need hardly be added that sterilization is to be advised without hesitation in the aforementioned cases of "complicated" congenital cataract. However, the reader definitely denied the validity of Fleischer's dictum that in all cases of congenital cataracts we must expect an unsatisfactory visual result from operative intervention. He referred to a recent publication of Heine (of Kiel) reporting the subsequent life history of subjects with congenital cataract, in which the author pointed out definitely how many persons are found to be quite capable of following various trades or professions after a correctly performed operation and how important it is to consider and judge each case of congenital cataract individually, with the greatest care and with regard for all the conditions. The reader's own experience tallied with that of Heine, and personal communications from Clausen, Fleischer and others have convinced him that a general consensus has been reached and that sterilization is not to be advised in many cases of zonular cataract, especially in cases of small opacities of limited extent which do not markedly interfere with sight and in which complications are entirely lacking, even if heredity has been proved.

The responsible adviser of the court will always have to ask himself to consider seriously the question whether in such cases families might not be extirpated who, after correction of a comparatively slight lesion, could be of service to their country. We must bear in mind that it is not wise to go beyond the limit in cutting down quantity in favor of quality.

RAPIDLY PROGRESSIVE PIGMENTARY DEGENERATION OF THE RETINA.
DR. STOCK, Tübingen.

A man of 25 was perfectly healthy until one and a half years previously. Within this space of time he became hemeralopic; then he showed a concentric contraction of the visual field, which progressed rapidly. The patient became blind six weeks before death. It was not the macula but an eccentrically located area in the temporal field which preserved its function longest. Clinically the patient presented the picture of retinitis pigmentosa, except that the pathologic changes were much grosser than usual. At first glance the fundus picture appeared to be that of an old, severe choroiditis.

However, with the binocular opththalmoscope and in the upright image it could be definitely noted that the foci had been produced by partial disappearance of pigment, partly by its heaping up in irregular clusters (*Klumpen*) and also by its emigration into the retina. While the patient was under clinical observation, extremely minute hemorrhages occurred in the layer of rods and cones and in the pigment epithelium, which later disappeared.

Histologically the process was one of decay of all the retinal nervous elements, beginning with the rods and cones, but almost at the same time involving the bipolar and ganglion cell layer in destruction. This degenerative process took place so rapidly that there was hardly time for proliferation of glial or connective tissue, so that vacuoles filled with fluid formed in the substance of the retina. The optic nerve still showed fairly good staining of the medullary sheath. In the brain and spinal cord almost all the ganglion cells, especially those of the cortex, had practically disappeared or were in the course of degeneration. In the meninges there were a few minute hemorrhages. Round cells were observed encircling some vessels of the brain and of the eye. As the patient was otherwise perfectly well and the postmortem examination failed to reveal any pathologic lesions in the viscera, it is impossible to venture even a surmise as to the cause of this acute degeneration of the nervous elements of the eye and central nervous system.

DR. BARTELS, Dortmund: Judging from the literature of our times and also from the discussions and opinions expressed at this meeting, one gets the impression that nowadays we do not consider only the disease in deciding our question but also what the patient can accomplish with or in spite of his disease. The law instructs that children whose vision does not permit them to follow the course of instruction at the ordinary schools and who have to be referred to a school for the blind are to be considered as sightless and if they are the subjects of hereditary disease mut be sterilized. But here, too, we have to take into consideration the feeblesighted. The schools for this class, it must be remembered, owe their origin directly to the fact that children were sent to schools for the blind when they were not "blind" at all, but merely feeblesighted. However, they are quite capable of following the entire school course if special methods are employed. Accordingly, they cannot be considered as blind. Those feeblesighted children who have a hereditary condition do not need to be reported without any further consideration, but should be reported only in case it appears that this condition has already been noted to be in the ascendancy. It might be well to take notice of what can be accomplished in a family

with congenital cataract. Incidentally, we note that in the schools for the feeblesighted there are many subjects with extremely high degrees of myopia and that just these children are unusually intelligent. The reader protested against the sterilization of male subjects with Leber's atrophy of the optic nerve on the ground of its being unnecessary. These patients never have diseased offspring. It is a great rarity for women to be affected with this disease, and those who have it can be sterilized. It is advisable, in order to avoid mistakes, to have each patient with such a condition examined independently by two ophthalmologists. Furthermore, the reader insisted that the judge of final instance, or the referee (*Oberbegutachter*) should render his decision not only on the basis of the documents in the case but after personal examination as well.

DR. LÖHLEIN, Berlin: There is no law which so well deserves to be called a humane statute, if judged by its meaning and purpose, as the law to prevent hereditarily diseased progeny. For it intends to prevent and to spare the individual, the family and the people as a whole a flood of the greatest sorrow by taking preventive action in advance against the destructive action of hereditary disease. It is true that the right, which, in agreement with the traditional wish of the medical profession, this law places in the hands of the physician, carries with it a great and serious responsibility, and we cannot subscribe to the opinion that the mere diagnosis of a hereditary malady alone would suffice for a decision to sterilize. On the contrary, the law requires also, as far as ophthalmology is concerned, that a condition of hereditary blindness or of serious developmental defect of the eye be present. The reader expressed the conviction that his colleagues all feel, as he does, the great responsibility of the ophthalmologist as an expert and that they follow the same principle, viz., limitation of their decision to unequivocal cases in which the necessity for the procedure can be made evident to the comprehension of an intelligent layman. We all have the standpoint that it is better at times to do too little than at any time to recommend sterilization lightly. Besides, it is the principle of the reader, as it surely is that of other conscientious experts, always to enlighten the patient and to convince him to the point where he himself, as far as possible, makes the request for sterilization and does not retain the feeling that he has been subjected to the coercion of a strict law. He will then consider his resolution, however hard it was for him to make, as his own liberating act. When we have spread the understanding of these requirements, step by step, in ever wider circles we may also hope to carry out successfully a task which is just as important, namely, in our capacity as advising physicians to have our purely humane counsel as to the self-denial of progeny followed in those cases which do not come under the provision of the law and in which a serious hereditary condition gives reason to anticipate danger but in which there is no actual blindness. The reader had in mind families with summation of cases of high myopia and detachment; the sisters of patients with Leber's disease, and similar cases. He stated that he was not in accord with the view that we put too high an estimate on what an individual can accomplish with energy and will, even if his visual remnant is, if taken by itself, insufficient. If he is the carrier of a serious hereditary malady and has a defect of vision which would

mark the average individual as practically blind, he should draw the logical conclusion, for it is hardly to be expected that the same unusual energy and will-power in his descendants will help them over the same serious defect. Several colleagues here have expressed a wish to have short directions drawn up and worked out which the ophthalmologist could follow in giving his expert findings and individual recommendation. The reader thinks it would not be right to have printed regulations planned which would lead the physician engaged in a case to look up the requirements in a perfunctory way and give his decision offhand as a sort of rubber stamp. The problem is one which is to be decided in each case, individually, and carries great responsibility with it. It takes time and labor. But one is dealing with matters of such unusual moment and serious importance that the expert should not be put in the position of having his well based, carefully considered and proved point of view superseded by any purely technical rules and regulations. We naturally aim to exchange the data of our experience and observations and so give facility to the judging attitude and uniformity to the method of arriving at a decision for each consultant. A valuable service is being rendered to such physicians in our broad, thorough and intensive discussions here of the question in its widest bearings, by the new edition of the commentary on the sterilization law, and by the publication of a handbook of hereditary disease, which is being prepared. It is important that we as experts should clarify the points in each individual case thoroughly, and, having done so, we should strongly urge that our expert judgment should receive full and due consideration. The reader is convinced that the difficulties which in this respect were at times presented in the past from the juristic side would tend to disappear as the jurist gained insight into the complexity of the conditions as to which the expert has to testify and as to which he alone can decide.

Dr. Meesmann, Kiel: Allusion was made to papers to be presented within the following days of the meeting by Holtz and Meesmann, which also deal with the subject of zonular cataract. It is improbable that rachitis, per se, leads to zonular cataract. There is required, in addition, some lowered function of the epithelial bodies. As this condition can be effectively treated today, according to Holtz, it is important to establish contacts with families in which zonular cataract is common, so that the women, particularly, can be treated during pregnancy with an ergosterol preparation.

Dr. von Bahr, Upsala, Sweden: Both Bücklers and Meesmann reject rachitis as a cause of zonular cataract. The reader thinks this is incorrect, as is Bückler's rejection of tetany as the etiologic factor. There are, as a matter of fact, cases of zonular cataract in which the condition is hereditary, and the reader has collected from the literature records of upward of 30 families showing a dominant heredity. But of over 700 patients with zonular cataract, well over half had suffered from cramps in childhood. This could not have been coincidence. Many showed signs of rachitis, too. J. Kugelberg, in Upsala, collected the data in a follow-up of about 150 children who had been treated at the hospital for spasmophilia in infancy or childhood. Of these, about one third had lenticular opacities similar to those observed in cases of zonular cataract, such as isolated peripheral spokes (*Reiterchen*), while

in three cases there was a fully developed zonular cataract. After presenting these clinical observations the reader submitted the data of some of his own experimental research. One hundred and nine young rats in 23 series were fed a rachitogenic diet, and the presence of rachitis was established by the x-ray plates. Forty-four of these rats received the diet mentioned uninterruptedly. They did not show cataract. For the other 65 rachitic rats the diet was modified so that it contained more phosphates. Of these 65 rats, 48 showed lenticular opacities. The rachitic rats reacted to the change in diet with an increase of response to irritation by the galvanic current and in a number of cases with spontaneous cramps or tremor. The controls, which had been given vitamin D in addition to the special diet, showed a very slight excess of irritability but no cataract. Lenticular opacities appeared in the eyes of the rachitic rats following excess irritability, in the majority of cases within forty-eight hours, and in many, in fact, within twenty-four hours, after the change in diet. These opacities were at first subcapsular, but with the development of the lens they had the appearance of zonular cataract. This condition, then, is due not to rickets alone but to rachitic tetany, or spasmophilia.

DR. STOCK, Tübingen: In his report, Clausen stated that we are all agreed that neuro-epithelioma (glioma) of the retina belongs, under all circumstances, to the hereditary diseases, and the patients are, accordingly, subject to sterilization. Personally, I disagree. In 28 cases of glioma in patients operated on in our Tübingen clinic in the last year or two we have had careful studies instituted and in only a single case found that an eye had been lost in the ascendancy. And even in this case it was not definitely determined whether the loss of the eye was due to glioma. After all, we do not sterilize in cases of carcinoma or sarcoma, although they might count as hereditary affections as logically—or as illogically!

' DR. FLEISCHER, Erlangen: The reader declared himself generally in accord with the preceding speakers, especially with Clausen in his reference to the serious responsibility which a physician takes on himself when he makes the report to the official medical examiner, as required by law, of a case of hereditary disease or certifies as to such a condition before the tribunal concerned with genetic hygiene. However desirable, hard and fast rules of invariable and permanent application cannot be laid down in the present state of knowledge, either for notification or for the certification just mentioned, but only general suggestions and directions can be given. Each subject must be conscientiously examined and tested. On the other hand, the reporting of suspicious cases cannot be avoided if one considers one's duty to the law and the community. In just such cases it is the notification which brings the matter before a duly constituted board for thorough investigation of the family history. It goes without saying that tact rather than drastic methods should characterize the activities of the physician, the official medical examiner and the juristic board.

DR. HERTEL, Leipzig: As was already mentioned by Bartels, difficulties are raised in these matters by the fact that the term blindness, which the law demands as a prerequisite for sterilization, has not been accurately defined. In the courts, statutes, codes for insurance and / or workmen's compensation laws there are many different specifications of

blindness and, especially, of the limits of useful vision. This uncertainty makes it very difficult to decide whether a patient is to be reported for sterilization and may be used by the patient himself to raise objections. This difficulty is most marked if the hereditary condition is monocular. We have noted large unilateral colobomas, aniridia, anophthalmos, macular degeneration and so on as evident signs that the subject is the carrier of pathologic heritage and that, accordingly, sterilization should be demanded. But what are we to do if the fellow eye is perfectly sound and has good vision? The carrier is perfectly capable of carrying on a trade, and we would, in a way, be acting against rather, than in accordance with, the law if we were to cause his sterilization. If the visual function is to be considered as a factor in the law and its application, we must ask the authorities to give us a more exact definition of this concept.

Dr. COMBERG, Rostock: The reader has gained the impression that today's statements are of importance, as they complete and confirm what has been set down in the literature on the subject in the last year or two. It would be desirable to have some sort of standing medical board in Germany, composed of individual ophthalmologists, whose office it would be to test out the various questions and moot points continuously and currently, either in lectures or / and correspondence, so that a consensus might be obtained as to the large, directional ideas which would be competent in deciding in the individual case. It is necessary for the large majority of ophthalmologists who do not take part in the proceedings of the German Ophthalmological Society to have a more detailed and definite basis for determination and judgment in difficult cases.

Dr. MÜGGE, Eisleben: The question whether we are to sterilize or not undoubtedly confronts the practical ophthalmologist with a serious responsibility, and I do not wish to be understood as pleading in favor of exaggerated claims and suggestions for sterilization. But, in regard to glioma, I have been greatly surprised to hear the view expressed here today repeatedly that in the majority of cases the condition is not hereditary. In some of the cases it undoubtedly is hereditary, and this applies also to the 2 brothers mentioned by Clausen in his report and who were my patients. The misery which these 2 children brought on their family may be judged by the facts which I add here. Within the space of a year and a half, one of the brothers had to have both eyes, the other, one eye, enucleated. In both subjects exenteration of the orbit had to be performed, in spite of which finally there was extension to the other eye, requiring enucleation. Any one who has lived through an experience of this sort will rather advise sterilization in one case too many than in one case too few.

(To be continued)

Book Reviews

Ocular Therapeutics. By Sanford R. Gifford, M.D. Second edition. Price, $3.25. Pp. 341, with 60 illustrations. Philadelphia: Lea & Febiger, 1937.

The first edition of this book appeared in 1932 and contained 272 pages and 36 illustrations. The new edition has 69 more pages and 24 more illustrations than its predecessor. The headings of the chapters are unchanged, but a new chapter on diseases of the orbit has been added. The format is unchanged except that the author has used italics for directing attention to matters of fundamental importance in therapy. This is a definite asset.

Much new material (based on the work of Bellows) concerning the relative efficacy of cocaine, phenacaine hydrochloride, butyn, nupercaine, pontocaine (paranormobutylaminobenzoyldimethylamino-ethanol hydrochloride) and metycaine has been added to the chapter on anesthetics. Various methods of infiltration anesthesia are discussed more fully than in the earlier edition. Vitamins, which were scarcely mentioned in the first edition, are given 5 pages; thirty drugs have been added to the list of drugs most used in ophthalmology, and a convenient procedure for preparing buffered ophthalmic solutions is outlined briefly. One correction should be noted at this time. In the formula for Acid Buffer Solution No. 1, at the top of page 62, 12.4 Gm. of boric acid is to be used instead of 6.2 Gm. New material concerning diathermy and phototherapy has been added to the chapter on physical therapy.

The chapter on diseases of the cornea has new sections on the treatment of marginal ulcer, Mooren's ulcer, smallpox keratitis, superficial punctate keratitis, epithelial dystrophy, neuroparalytic keratitis and keratitis due to lagophthalmos.

The new chapter on diseases of the orbit is concerned with orbital cellulitis; thrombosis of the cavernous sinus; exophthalmos in association with hyperthyroidism; orbital hematoma; fracture of the wall of the orbit, and pulsating exophthalmos.

This new edition of "Ocular Therapeutics" is a sane, well balanced and conservative presentation of the various drugs, physical agents and mechanical procedures which the author has found to be of value. It deserves to be on the bookshelf of every ophthalmologist, and it should be required reading for residents in ophthalmic hospitals.

W. F DUGGAN.

Directory of Ophthalmologic Societies *

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66, Boulevard Saint-Michel, Paris (6e).
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

INTERNATIONAL OPHTHALMOLOGIC CONGRESS

Secretary: Dr. E. Marx, Costzeedijk 316 Rotterdam, Netherlands.
Place: Cairo. Time: Dec. 8-14, 1937.

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

President: Dr. A. F. MacCallan, 33, Welbeck St., London, W., England.

FOREIGN

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. P. E. H. Adams, 6, Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13, Mitchell Ave., Jesmond, Newcastle-on-Tyne.

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

GERMAN OPHTHALMOLOGICAL SOCIETY

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. T. E. Ashdown Carr, 34, Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3, Midan Soliman Pasha, Cairo.
Time: March 1938.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. Gordon M. Holmes, 9, Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27, Weymouth St., London, W. 1.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 8-10, 1937.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.
Place: Lindley'a 4, Warszawa.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31, East Southernhay, Exeter, England.
Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm, Sweden.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: San Francisco. Time: June 13-17, 1938.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Lee W. Dean, Washington University Medical School, St. Louis.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

American Ophthalmological Society

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

New England Ophthalmological Society

President: Dr. James J. Regan, 520 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Road, Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. A. J. Ridges, Walker Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Salt Lake City. Time: May 24-27, 1937.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. W. Howe, 740 St. Helens Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501 Seventh St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Robert Griswell, 707 Washington Ave., Bay City, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. L. H. Hohf, Yankton, S. D.
Secretary-Treasurer: Dr. J. C. Decker, Francis Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.
Place: New Orleans. Time: Nov. 30-Dec. 1-3, 1937.

Southwestern Michigan Triological Society

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. C. W. Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 227 16th St., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Walter L. Hogan, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. B. H. Minchew, 701 Elizabeth St., Waycross, Ga.
Secretary-Treasurer: Dr. Edward S. Wright, 1001 Medical Arts Bldg., Atlanta, Ga.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. R. Dillinger, French Lick.
Secretary: Dr. Frederick V. Overman, 705 Hume-Mansure Bldg., Indianapolis.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. James A. Downing, 406 Sixth Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.
Secretary: Dr. D. R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. C. Coulter Charlton, 124 S. Illinois Ave., Atlantic City.
Secretary: Dr. H. L. Harley, 124 S. Indiana Ave., Atlantic City.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Walter S. Atkinson, 168 Sterling St., Watertown.
Secretary: Dr. Marvin F. Jones, 121 E. 60th St., New York City.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. Rosenberger, 221 Fifth St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514 Sixth St., Valley City.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. Nathan Bolotow, 108 Waterman St., Providence.
Secretary-Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. R. E. Houston, 103 E. North St., Greenville.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. W. W. Potter, 601 Walnut St., Knoxville.
Secretary-Treasurer: Dr. W. D. Stinson, 248 Madison Ave., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Edwin W. Burton, University of Virginia, University.
Secretary-Treasurer: Dr. George G. Hankins, 202 Medical Arts Bldg., Newport News.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. F. O. Marple, First Huntington National Bank Bldg., Huntington.
Secretary: Dr. J. E. Blaydes, First National Bank, Bluefield.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. Samuel T. Hubbard, 294 State St., Hackensack, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, First-Central Tower, Akron.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Jesse W. Downey Jr., 529 N. Charles St., Baltimore.
Secretary: Dr. Mary L. Small, 18 W. Read St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third
Thursday in February, April, May, October and December.

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Henry Mundt, 30 N. Michigan Ave., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third
Monday of each month from October to May.

CINCINNATI OPHTHALMIC CLUB

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15
p. m., third Monday of each month except June, July and August.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. A. D. Ruedemann, 2020 E. 93d St., Cleveland.
Secretary: Dr. Fred W. Dixon, 1029 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. A. B. Bruner, 629 Euclid Ave., Cleveland.
Secretary: Dr. M. W. Jacoby, Hanna Bldg., Cleveland.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. Andrew Timberman, 21 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Claude S. Perry, 40 S. Third St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. A. W. Davidson, City National Bank Bldg., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 720 Medical-Professional Bldg., Corpus Christi,
Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Hugh L. McLaurin, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Maxwell Thomas, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. A. W. Greene, 148 Barrett St., Schenectady.
Secretary-Treasurer: Dr. Joseph L. Holohan, 317 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 500 Metz Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Henry C. Haden, 1914 Travis St., Houston, Texas.
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. C. Daniel, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. H. B. Davis, 1101 Grand Ave., Kansas City, Mo.
Secretary: Dr. Byron Black, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach, Calif.

Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.

Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.

Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.

Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.

Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.

Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.

Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.

Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.

Secretary: Dr. R. O. Hychener, 130 Madison Ave., Memphis, Tenn.

Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.

Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.

Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.

Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.

Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.

Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.

Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.

Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.

Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. John H. Dunnington, 30 W. 59th St., New York.
Secretary: Dr. LeGrand H. Hardy, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. W. H. Stokes, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Falk Clinic. Time: 4 p. m., second Friday of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. John T. Crebbin, 624 Travis St., Shreveport, La.
Secretary-Treasurer: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. A. Veasey Jr., 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Philip B. Green, Old National Bank Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto, Canada.
Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 Eye St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

TREATMENT OF DETACHMENT OF THE RETINA

H. ARRUGA, M.D.

BARCELONA, SPAIN

The surgical treatment of detachment of the retina is undoubtedly the most interesting subject in ophthalmology today. It is to Gonin, the celebrated professor in Lausanne, that ophthalmologists owe the discovery of this treatment. He studied the condition for many years, and eventually his efforts were crowned with success. The importance of Gonin's discovery is enormous. The fact that about 20 per cent of the adult blind lose their vision through detachment of the retina, that this number was formerly practically incurable and that now through operation the trouble can be cured in the great majority of cases surely confirms the importance of the new treatment. I cannot speak of this subject without offering homage to this investigator, who, unfortunately, died two years ago.

Gonin was learned, sincere and good. Learned because he arrived at his discoveries not by hazard but as the result of prolonged study and many investigations. Sincere because he never wrote anything but the truth and wrote without the slightest exaggeration. This is possibly the reason why at the time of his first publications no great attention was given to them. The 20 to 25 per cent of favorable results which he then modestly noted have been proclaimed by other authors with other methods. Finally, Gonin was good. All those who knew the man intimately knew how he lived for his family, his friends and his patients. A last example was the bestowing of a great part of his modest fortune on the blind of Lausanne.

The subject is large, but so much of it is now well understood that I can limit myself to a brief article. I have found in traveling and in reading foreign articles that the subject is still difficult for many and frequently not well understood. To speak truthfully, the treatment of detachment of the retina has not been adopted as well as it should be. To my mind, the principal reason is that, owing to the diversity of clinical types, the operator must adopt operative measures according to the condition in each case. The majority of ophthalmologists perform a good operation for cataract or glaucoma, in which the procedure is gen-

erally the same in all cases, but in operations requiring various technics, such as dacryostectomy or operation for detachment of the retina, conditions are more difficult, and in atypical cases the operator is handicapped by lack of experience. As operation is really easy in the cases in which the condition is uncomplicated, it is necessary to begin by operating in this type of case and not hesitate, because success will encourage the confrères to operate with greater success in the more difficult cases. It is preferable, from a humanitarian point of view, that the majority of ophthalmologists operate with success in 50 per cent of the cases rather than that certain ones obtain good results in 70 per cent. The first group passes with time into the second.

The greatest difficulty lies in the ophthalmoscopic examination, for the procedure of operation depends on careful examination of the eyegrounds and on the ophthalmoscopic control during operation. When it is difficult to examine the eyeground on account of opacities, the prognosis is poorer, as the study of the eyeground and the retina is interfered with. Many of the colleagues are content with the results obtained by the relatively simple procedure (extensive operation in the suspected region, particularly large barrages). They obtain with this technic cures in 40 to 50 per cent of the cases, which, when compared with the results obtained by subconjunctival injections of sodium chloride, is, of course, marvelous. But if the colleagues will attempt to make a more careful localization of the area to be operated on, they will be able to improve their results in from 20 to 25 per cent of the cases.

Of course, a large barrage and an extensive operation are frequently indispensable, but it is important to try to do the operation with the least danger to the eye, as the intolerance on the part of the eye increases with the extent of the operation, and, moreover, it is desirable to sacrifice the smallest possible area of retinal territory. While it is not always possible to localize the lesion carefully, an attempt must be made with all the skill at one's disposal to obtain an accurate localization. This requires a prolonged and often fatiguing ophthalmoscopic examination. It is much easier not to wear oneself out in the dark room and do a large and not necessarily limited operation, but this is not so good for the patient.

OPHTHALMOSCOPIC EXAMINATION

Ophthalmoscopic examination should be made with direct and indirect images, with maximal dilatation of the pupil produced by instillation of atropine sulfate or epinephrine hydrochloride or by a subconjunctival injection of epinephrine hydrochloride. The indirect image requires a very intense light. The best source is the projection

lamp with the use of from 100 to 200 volts. In this lamp the filaments are zigzag and placed in the same plane. The glass may be either dull or clear. The indirect image is particularly necessary in examination of the extreme periphery of the retina. For the direct image most of the electric ophthalmoscopes are good. They must carry a correction up to 30 D. and have a good intensity of light. The direct image is indispensable for judging differences in level and in the definition of small details. The result of the ophthalmoscopic examination must be entered on a schematic drawing designed according to that of Amsler and Dubois. There is no other method of control for a good study of the case or in order to acquire experience. I have occasionally been called to operate on a patient with a good history, the description of whose symptoms and pathologic observations took up from eight to ten typewritten pages, but in whose case there had been no attempt to make a drawing. In other words, the most important thing remained to be done. I rapidly read the text, principally out of politeness, and then set about making a drawing. The necessity of a good drawing is important not only to serve as a guide during the operation but for postoperative control and to enable one to judge the results in general when the different cases are studied in groups according to the clinical character of the condition as shown by the drawing. A good drawing helps to determine the proper method of treatment and operation and is also an aid to prognosis and in acquiring experience. Ophthalmoscopic control makes the operation more effective and perfects one in the technic, as it is a help always to see what one is doing. Sometimes the postoperative results are surprising. One may expect a good result, and it does not take place. In other cases, a condition may seem difficult to cure, and the result of operation proves to be good. These surprises would be less startling if one had made a careful ophthalmoscopic examination during and immediately after the operation for the purpose of control.

When one sees that the electrodes have not touched the retina and, after the subretinal liquid has been allowed to escape, that the retina still remains far from the choroid and that folds in the retina are still present, the prognosis is not favorable.

In order to show the importance of ophthalmoscopic examination I shall report two unusual cases. In one of these the patient was a woman presenting a macular hole. While I was making a diathermic surface coagulation I coagulated at the same time the blood in the superior retinal vein, and on examining the eyeground was startled to see a thrombosis. The vision was lost in a sector of the field as the result of this vascular lesion.

In another case, in which there was a tear in the upper part of the eyeground, with bulging of the retina which had not become reduced

by rest in bed, an attempt was made with the long diathermic needle to grasp the retina and draw it toward the choroid. I then found with the ophthalmoscope that I had torn the retina from the place where the needle had touched it as far as the posterior pole near the optic nerve. Without ophthalmoscopic examination I should not have been able to understand the reason for my ill success.

REPAIR OF HOLES

The meridian is noted by means of a mark made at the limbus with china ink or with an alcoholic solution of gentian violet by a fine needle. The distance of the hole from the limbus is more difficult to determine. One may estimate the distance in papillary diameters, as has been done for a long time, but it is much better to orientate oneself with the ophthalmoscope after having made a galvanopuncture, or, better, a diathermic puncture or after having introduced a needle or a fine instrument into the eye, or with transillumination or diaphanoscopy. The old method of estimating the distance in papillary diameters between the ora serrata and the tear is not entirely exact, because the ora serrata is not always visible, and it is difficult to estimate the exact location of the ophthalmoscopic peripheral field. In most cases the distance in millimeters is measured with a compass on the sclera, and this is generally sufficient.

Thermopuncture with a fine galvanocautery may give good orientation, but it has the inconvenience of sometimes making a hole in the retina, the choroid and the sclera, which drains the subretinal cavity excessively.

Diathermic punctures are of great importance when the retina is not too bulging. The diathermic needle is introduced into the suspected region, and if one does not recognize with the ophthalmoscope any change in the retina in the form of a white patch some of the subretinal fluid is allowed to escape with the aid of pressure exerted on the eyeball. The needle is again introduced at the marked area, and ophthalmoscopic examination is repeated. Then in nearly all cases the white area will be discovered, and its relation to the tear can be determined.

If the retina shows bulging and this does not improve after the escape of the subretinal liquid so that the diathermic puncture is not visible, a small needle or knife may be introduced, which is more easily visible with the ophthalmoscope. Transillumination consists in throwing light on the tear with the ophthalmoscope. The assistant notes which part of the sclera is illuminated. When the operator sees that the tear is well illuminated, the assistant makes an electric puncture at this part of the sclera or applies china ink or gentian violet. In diaphanoscopy, on the other hand, a transilluminator, such as the lamp of Lange or

that of Rochon and Duvigneaud, is used. The sclera is illuminated while the operator is examining the eyeground, and when he perceives the light passing through the suspected area the assistant is asked to make a mark, or to pass the current, if the lighting apparatus carries the diathermic electrode as suggested by Strampelli.

Whenever the condition is simple, any of these procedures is good; but if the retina is very bulging and the vitreous is cloudy, the difficulties increase, and the operation is accordingly more difficult. In any case, all efforts to obtain good localization will be recompensed by better results of treatment.

ANESTHESIA

A local anesthetic is sufficient in most cases, and it is well to administer hypnotics. Instillations of an anesthetic should not be too frequent, in order not to change the corneal surface. The eye, of course, must be closed between instillations. The injection should be made into Tenon's capsule and not subconjunctivally.

OPERATION

Cautery.—The method of Gonin is useful in cases of very simple conditions in which it is possible to make an accurate localization so that the diathermic and perforating applications will fall just in the place of the tear. This method has been practically abandoned today on account of the danger of hemorrhage, which sometimes complicates the postoperative course.

Caustics.—The use of caustics is efficacious but very difficult, as it is hard to trephine the sclera without perforating the choroid. It is much more painful than other procedures, and one obtains as good results with other methods, especially diathermy. This procedure was suggested by Lindner and Guist.

Diathermy.—Diathermy is the most extensively used method at the present time. First designed by Weve, it was rapidly adopted by most operators on account of its freedom from danger. Diathermy may be applied in two ways—to the surface or perforating the surface. To apply surface coagulation one must use an instrument with a flat or a round tip, and the sclera then acquires a slightly grayish color. To obtain this result a current of from 30 to 60 milliamperes is necessary. Perforating diathermy requires a current of greater intensity—from 80 to 150 milliamperes, according to the condition and the size of the electrode. Each method has its definite indications. In general, one uses both methods. When the detachment is flat and the retina is not far from the choroid, surface diathermy followed by a small perforation to drain the subretinal fluid is the best method. In cases of disinsertion

near the ora serrata this method is usually satisfactory. In cases of macular holes it is prudent to use surface diathermy in the macular region very cautiously so as not to injure the retina.

In cases in which there is a large, bulging detachment, surface diathermy may be used associated with perforating diathermy to obtain drainage of the subretinal spaces. This drainage must be as much as the retroretinal cavity requires. An excessive bulging of the retina is an inconvenience; an attempt is made to reduce it before the operation by rest in bed and during the operation by compression of the eyeball after puncture of the sclera. In using perforating electrodes one may add a kind of surface diathermy without actually perforating the sclera. This is suitable if it is difficult to obtain dry surface areas, because for surface diathermy to be effective the sclera must not be moist.

In making diathermic punctures one should not make these too close to one another, as scars will result. It is always best to leave a space of 0.75 mm. between each puncture. This, of course, applies only to perforating diathermy. In surface coagulation the punctures may be made closer together. Vorticose veins should also be carefully avoided, experience having shown that their obstruction by coagulation retards healing after operation, notwithstanding the fact that the vessels in the choroid present many anastomoses.

The continuous barrages, when applied transversely, that is, parallel to the equator of the eye, have the inconvenience of coagulating the vessels in an ocular sector and obstructing the circulation for some time; they possibly also interfere with the function of the ciliary nerves. This is probably the reason why in cases in which the coagulation has been extensive there is difficulty in dilating the pupil. It is important that the drainage of the subretinal pocket exist long enough, because if the perforations close too early, before the liquid has been absorbed, the retina will not come into contact with the choroid, and adhesion will not take place. To insure satisfactory drainage the openings must be sufficiently large. The fine needles used in diathermy often close too rapidly and are applicable in cases in which the retina is only slightly detached. If there is considerable fluid the perforations should be made with a needle 0.5 mm. in thickness, or, when a fine needle is used, the perforating action should be prolonged and the borders of the hole enlarged. Galvanocautery may also be used to make a perforation for drainage or a trephine without endangering the choroid. The latter structure is then perforated with a blunt instrument, such as a strabismus hook. It is very necessary (and in this lies the secret of success) that the retina should be in contact with the choroid at the end of the operation. Nothing will produce adhesive choroiditis if the two membranes remain separated by a layer of subretinal liquid instead of being in

contact or slightly separated by an exudate produced at operation. If the galvanopuncture is going to be used for drainage, it is well to make a circle of diathermic applications first so that the vessels are coagulated. If the diathermic punctures are made where the surface coagulation has been previously done the sclera will be found much thickened. Personal experience, as well as seeing colleagues operate, helps one in judging the suitable degree of intensity for diathermy. The milliamperimetric measures are, of course, only relative. It is better to judge the effect by the change produced in the tissues, particularly with the ophthalmoscope. For surface coagulation, pyrometric electrodes are excellent. In cases of inferior or lateral detachment one may facilitate the adaptation of the retina by making an injection of air at the superior pole.

POSTOPERATIVE TREATMENT

When the operation is finished, atropine sulfate is instilled and a binocular bandage applied. The patient must remain in bed for several days, and it is always well, as Gonin has suggested, to incline the head of the patient to the side on which the operation has been performed. If the operation was done in the lower half of the retina the patient may be allowed to sit up partly. The length of time in bed depends on the condition. In general, from six to eight days are sufficient; in case of severe involvement this time may be lengthened, though it is more important to keep the eyes quiet than the body, because it is the movement of the eyes which causes trouble for the retina. It is best not to uncover the eye which has not been operated on too soon, as movement of the eyes will interfere with the healing of the retina. Stenopeic spectacles may limit the movements of the eyes, but the binocular bandage is much more efficacious. The dressing should be changed every three days and atropine sulfate instilled. On the sixth day the eyeground may be examined, and if progress is favorable and the condition is not severe the patient may be given some liberty. It is usually not wise to remove the binocular bandage before twelve days. If it is necessary to repeat the operation it can be done after this time. In general, I permit my patients to leave off their bandage and to wear stenopeic glasses after twelve to twenty days, according to the condition.

PROGNOSIS

The prognosis of detachment of the retina depends on the clinical variety of the condition in each case. In simple cases in which there are small holes, without a great degree of detachment and in which the detachment is of only a few days' standing, recovery is the rule. In cases of detachment with a tear at the ora serrata the condition is always easy to cure, but the extent of the detachment, the number of holes, the

duration and, particularly, disturbance of the vitreous make the condition difficult to cure in proportion to the development of any of these factors.

Myopia does not play a large rôle, though in cases in which the retina is very atrophic there is a greater tendency to recurrence. The age and general state of health of the patient also are important. Finally, the process of absorption and cicatrization will depend on the health of the eye.

PATHOGENESIS

A new problem which the interest in the modern treatment of detachment of the retina has created is that of the pathogenesis. The rôle of the retinal holes in the development of a detachment is incontestable; but the difficulty is to interpret how these perforations act. A number of theories have been advanced, but they do not hold for all cases. Undoubtedly there are several causes for detachment of the retina. I refer not to the cases of detachment without holes following exudative choroiditis, but to the great majority of cases, in which there are holes. In these cases the pathogenesis or the process of formation of the holes may proceed in one of two ways: by adhesion of the retina to the vitreous and detachment on movements of the eyeball or by adhesion of the retina to the choroid and choroidal exudation; the latter is much less frequent than the former. Pathologic examination of eyes with recent detachment has shown that the retina is atrophic and is much altered in each case and that through succussion or movements of the eyeball the retina becomes detached. To my mind, the adhesions which exist between the framework of the vitreous and that of the retina are the agents which produce the tears. It is well known that inflammation of tissues, if it lasts for any length of time, leads to atrophy and fibrosis of these tissues and adhesions to the adjoining tissues. Anatomic examinations of eyes with recent detachment have shown atrophy of the retina in some cases and adhesions to the vitreous and particularly pigmentary inclusions in the retina in others, which demonstrates the preexistence of an inflammatory process in both membranes. The inflammatory process, however, must not be so intense that the retina becomes adherent to the vitreous and not to the choroid. In the latter case the retina would not become detached. This explains detachment in cases of old choroiditis in which the inflammation is mild (black areas in the eyeground) and its absence in cases in which there are white patches, for in the latter the inflammation has been intense and has even destroyed the choroid, so that the two membranes have become adherent. There is a simple experiment which will demonstrate the ease with which the vitreous will adhere to the retina when the latter membrane is inflamed. After removal of the conjunctiva in a rabbit and irritation of the sclera at the level of the equator with either chemical or physical agents, if

the eye is removed after several days or weeks and placed in solution of formaldehyde U. S. P. and then in alcohol, the vitreous retracts as a result of dehydration. One can then see that the vitreous is much retracted but remains adherent to the retina at the place of irritation. I think that the adhesion between the vitreous and the retina develops in this way, and then a succussion or movement of the eye detaches a piece of the retina. On movements of the eyeball, more and more vitreous passes behind the retina. There may be tears produced by choroidal exudation. There must be a previous adhesion between the retina and the choroid to produce a rupture of the retina by raising of the retina in the periphery. This is, however, a much rarer phenomenon than the former.

CONCLUSION

In conclusion I cannot do better than repeat what I said at the beginning of this article and express my gratitude and render homage to Gonin, that benefactor of mankind.

TREATMENT OF OCULAR TUBERCULOSIS

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The results of treatment with tuberculin in 42 cases of ocular tuberculosis in the Wilmer Ophthalmological Institute were analyzed and reported in 1928.[1] The statement was then made that the manner in which tuberculin acted, whether as a specific desensitizing agent or by augmenting the specific local resistance by a succession of small stimuli, was not known. Since this report our knowledge of the influence of allergy and immunity on tuberculous lesions has been considerably clarified. We have therefore reviewed the cases of all patients with ocular tuberculosis treated with tuberculin or by other procedures in the public wards or in private practice in the Wilmer Ophthalmological Institute and have analyzed these cases in the light of this newer knowledge of tuberculosis. The study is the subject of this report.

The influence of allergy and immunity on local tuberculous lesions has already been discussed by one of us [2] elsewhere and will be further elaborated in the reports of general studies now in progress. For our purposes here the subject may be summarized as follows:

INFLUENCE OF ALLERGY AND IMMUNITY ON TUBERCULOUS LESIONS

Tuberculous lesions may vary between the acute inflammatory caseating lesions seen in highly allergic animals, the slowly progressive lesions of the normal animal and the rapidly limited, quickly encapsulated lesions in the highly immune animal. This wide difference is seen also in the lesions of ocular tuberculosis. On the other hand, there are the acute caseating necrotizing lesions with a tendency to perforate the globe, and at the other extreme, the self-limiting lesions which

1. Woods, A. C., and Rones, B.: The Therapeutic Use of Tuberculin in Ocular Tuberculosis, South. M. J. **21**:613, 1928.

2. Woods, A. C.: Allergy and Immunity in Ophthalmology, Baltimore, Johns Hopkins Press, 1933.

heal but have a tendency to recur. Experimental studies in both general and ocular tuberculosis have shown that with similar dosage and virulence of the tubercle bacilli this difference in the lesions is due to the underlying hypersensitivity and immunity of the infected animal.

Since the time of Koch it has been recognized that normal and tuberculous animals react differently to tubercle bacilli. Under parallel conditions, the normal animal reacts with the formation of slowly developing tuberculosis with tubercle formation, while the tuberculous animal shows an acute localized inflammatory reaction, inhibition of dissemination and some actual destruction of the invading bacilli. The acute inflammatory reaction has long and correctly been recognized as due to hypersensitivity of the tissues of the tuberculous animal. The tuberculous animal becomes allergic to tuberculoprotein. It was believed for many years that the inflammatory allergic reaction was responsible for the resistance to further infection shown by the tuberculous animal and that this reaction fixed the bacilli at the site of injection, prevented their dissemination and aided in their encapsulation and destruction. Under this conception, allergy was responsible for immunity. In 1929 Rich and McCordock[3] sharply challenged this view and showed there was no experimental evidence for its support. In a series of experiments they demonstrated that allergy and immunity were two distinct entities, in no way related. Allergy was responsible for the inflammatory, exudative and necrotizing phases of tuberculous lesions, while immunity was responsible for the fixation of the bacilli and their encapsulation. Extending their researches to other infections, they showed that the allergy could be entirely removed by desensitization of the experimental animals, while the immunity was undisturbed. In 1933 Rothschild, Friedenwald and Bernstein[4] illustrated the same fact for tuberculosis.

The nature of immunity is not entirely clear; certainly there are two factors—a humoral and a cellular factor. The humoral element is concerned with the immune antibodies which tend to fix and immobilize the bacilli, while the cellular element is concerned chiefly with the macrophages which engulf and destroy the bacilli. Other factors, the immobilizing effects of the fibrin barrier and possibly certain physiochemical factors in the inflammatory lesions, may be concerned in immunity. Therefore, in the light of this knowledge that allergy incites

3. Rich, A. R., and McCordock, H. A.: An Enquiry Concerning the Rôle of Allergy, Immunity and Other Factors of Importance in the Pathogenesis of Human Tuberculosis, Bull. Johns Hopkins Hosp. 44:273, 1929. Rich, A. R.: The Demonstration that Allergic Inflammation Is Not Necessary for the Operation of Acquired Immunity, Proc. Nat. Acad. Sc. 16:460, 1930.

4. Rothschild, H.: Friedenwald, J. S., and Bernstein, C.: The Relation of Allergy to Immunity in Tuberculosis, Bull. Johns Hopkins Hosp. 54:232, 1934.

the spreading, destructive, inflammatory phases of the tuberculous reaction and immunity is responsible for the healing and encapsulation of the lesion, in the attack on a local tuberculous lesion one's primary duty is to remove the fatal hypersensitivity of tissue and promote immunity.

THERAPEUTIC ACTION OF TUBERCULIN

There are obviously two different conceptions of the mode of action of tuberculin on local tuberculous lesions. For want of better names we term these the "perifocal" and the "desensitization" concept. The perifocal concept is founded on the old view that allergy is responsible for immunity and assumes that tuberculin excites repeated minor perifocal reactions about the local lesion, these repeated reactions producing local immunity which promotes encapsulation of the lesion and destruction of the bacilli. On the basis of this perifocal concept, tuberculin should be used in doses just beneath the point of focal reaction. Inactivity of the lesion is interpreted as the attainment of a local immunity, and, no further perifocal reactions being desired, tuberculin therapy is abandoned. The desensitization concept is founded on the idea that allergy is responsible for the inflammatory phases of the tuberculous lesion and is unrelated to immunity. Through removal of the fatal tissue hypersensitivity, which is responsible for the inflammatory and destructive phases of the tuberculous lesion, the bacilli are reduced to the status of inert foreign bodies, and the immunity acquired by the tuberculous infection, unrelated to allergy, effects their encapsulation and destruction. On the basis of this concept, tuberculin is used in small doses, especially in the presence of an active inflammation, and is continued in increasing doses over a long period after the lesion is apparently healed, the fundamental idea being to achieve and to maintain desensitization of tissue and to prevent the return of hypersensitivity which invariably follows the premature cessation of the use of tuberculin.

It is obvious that tuberculin used on the basis of the perifocal concept of action will have a definite desensitizing effect; in fact, more rapid desensitization may possibly be accomplished by giving tuberculin in quantities sufficient to provoke even minimal reactions. Therefore, whether tuberculin is given with the idea of producing perifocal reactions or with that of desensitizing the ocular tissues, primary desensitization of the same degree will probably ensue. This is the probable explanation for the temporary good effects of tuberculin reported by so many authors. The salient differences between the use of tuberculin according to the perifocal concept and that according to the desensitization concept are, first, the radically different views underlying its use and, second, the fact that under the perifocal concept the use of tuberculin is aban-

doned when inactivity of the lesion is obtained, while under the desensitization concept tuberculin must be used for long periods, and the dose increased steadily during periods of remission when the eye is quiet, to maintain desensitization which otherwise almost invariably would recur.

MATERIAL

The cases which form the basis of this report are, first, the 42 cases reported by Woods and Rones in 1928, in 4 of which the patients have been followed consistently since that date and treated on the basis of the desensitization concept of the use of tuberculin, and, second, 111 patients seen in the public ward and dispensary and 22 patients from private practice for all of whom the final diagnosis was ocular tuberculosis and who were treated with tuberculin in the Wilmer Ophthalmological Institute since 1926.

DIAGNOSIS

The diagnostic problem of ocular tuberculosis will be fully discussed in a later communication. For these patients the diagnosis was reached on the basis of the usual criteria: the history, the clinical picture of the diseased eyes, the tuberculin reaction of the individual patients and a thorough physical examination to determine the possibility of systemic tuberculosis and to eliminate other etiologic factors such as syphilis and focal infection. A Wassermann test and special examinations of the teeth, tonsils, accessory nasal sinuses and genito-urinary system were done as a routine. Special bacteriologic examinations and sensitivity tests were done when indicated. All foci of infection, such as periapically infected teeth, infected tonsils or sinuses and infections of the genito-urinary tract, were eradicated as far as possible. The tentative diagnosis of ocular tuberculosis was made final only after the effect of removal of foci of infection had been observed and it was clear the removal of such foci had no effect on the ocular lesion and did not appear casually connected with it.

The most pertinent point in the diagnosis was the tuberculin reactions of these patients. The Mantoux or intracutaneous test with graded amounts of tuberculin, 0.001, 0.01 and 0.1 mg., was done as a routine. On account of the danger of focal reaction the subcutaneous and the Calmette test were never used, and the von Pirquet test was likewise not employed on account of its extreme sensitiveness. In addition to the 175 patients who form the basis of this report, there were 5 additional patients with ocular tuberculosis not treated with tuberculin and 10 patients with ocular tuberculosis proved by histologic examination of the enucleated eyes [5] for whom we have records of the tuberculin reac-

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5. These are the 10 patients previously studied by Friedenwald and Dessoff.

tions. The results of the Mantoux test in these 180 clinical cases of ocular tuberculosis and the 10 cases of histologically proved tuberculosis are shown in table 1.

Of the 180 patients with clinical ocular tuberculosis, 96, or 53.4 per cent, reacted to 0.001 mg. of tuberculin; 75, or 41.6 per cent, had cutaneous reactions only to 0.01 mg., and 9, or 5 per cent, reacted only to 0.1 or 1 mg., or were insensitive. Somewhat similar figures were shown for the group of 10 patients with histologically proved ocular tuberculosis; 6, or 60 per cent, had cutaneous reactions to 0.001 mg.; 2, or 20 per cent, to 0.01 mg., and 2, or 20 per cent, to 0.1 mg. or less. These figures bear out the findings of Hart,[6] who tested the cutaneous reactivity of 1,030 patients with active systemic tuberculosis. He found that 13 per cent gave entirely negative reactions to 0.01 mg., and on testing with stronger doses he found that even with 100 mg. there was a residual error of 2.2 per cent in the test. These figures illustrate well the fallacy

TABLE 1.—*Hypersensitivity to Tuberculin of Patients with Ocular Tuberculosis*

Condition	Number	Sensitive to 0.001 Mg.		Sensitive to 0.01 Mg.		Sensitive Only to 0.1 or 1 Mg., or Insensitive	
		Number	Percentage	Number	Percentage	Number	Percentage
Clinical ocular tuberculosis.................	180	96	53.4	75	41.6	9	5
Histologically proved ocular tuberculosis.....	10	6	60.0	2	20.0	2	20

of the view that a high degree of cutaneous reactivity should be expected in patients with ocular tuberculosis and that a low degree of cutaneous reactivity is positive evidence against the presence of ocular tuberculosis. Unfortunately, one has no means of determining the degree of the ocular sensitivity, no knowledge of the influence of local disease on the ocular sensitivity and no information on the relationship of cutaneous and ocular sensitivity in patients with ocular tuberculosis.

METHODS AND RESULTS OF TREATMENT

In addition to tuberculin treatment, these patients all received such general hygienic treatment as was compatible with their social status. Certain selected patients were given special treatment, paracentesis, autohemotherapy, phototherapy and subconjunctival injections, as indicated. The results of these therapeutic measures, the use of tuberculin, general hygienic treatment and special treatment, will be discussed in order.

6. Hart, P. D.: The Value of Tuberculin Tests in Man, Medical Research Council, Special Report Series, no. 164, London, His Majesty's Stationery Office, 1932.

Treatment with Tuberculin.—Tuberculin, whether given on the basis of the perifocal concept or that of the desensitization concept, was at the onset of treatment administered in the same manner outlined in a previous communication. The initial dose was usually 0.0001 mg. of Denys bouillon filtrate, made and standardized in the laboratories of the Wilmer Ophthalmological Institute. The doses were increased in the usual manner and repeated every four days until a dose of 0.9 mg. was given. Thereafter injections were given once a week. The site of injection was watched for local reactions, and the eyes were constantly observed for any suggestion of focal reactions. With the occurrence of local reactions the dose was dropped three or four levels, and with any suggestion of focal reactions it was dropped drastically—eight levels—and increased only when local or focal reactions had disappeared, the effort being made consistently to keep the dose below the patient's individual point of reactivity.

TABLE 2.—*Results of Tuberculin Therapy in Patients Receiving Only One Course of Tuberculin*

Ocular Involvement	Number of Cases	Results		
		Healed	Improved	Unimproved
Keratitis......................	23	10	12	1
Kerato-iritis...................	17	6	7	4
Scleritis......................	7	5	0	2
Posterior ocular tuberculosis..	62	35	16	11
General uveitis................	34	18	9	7
Total.....................	143	74	44	25
Percentage................	100	51.9	30.7	17.4

The most important difference in treatment between that based on what we have termed the perifocal concept and that based on what we have called the desensitization concept lies in the duration of treatment. The majority of these patients, 143 in all, received only one course of tuberculin. At the completion of the course, when the patient had received 90 mg. of tuberculin without reaction, treatment was abandoned. In the greater number of these patients the decision to abandon treatment was influenced by satisfaction with the clinical outcome or by conviction that tuberculin had accomplished all that could be expected. In other patients, for various reasons, it was impossible to continue treatment. The results in these patients, whose treatment with tuberculin falls under what we termed the perifocal concept of action, are shown in table 2. Seventy-four, or 51.9 per cent, showed apparent healing of the ocular lesions; 44, or 30.7 per cent, showed definite clinical improvement, while 25, or 17.4 per cent, were unimproved.

The method of treatment was radically different for the 32 patients for whom tuberculin was used on the basis of the desensitization concept

of action. After the dose of 90 mg. or the maximum dose that the patient could safely take was attained, injections were continued steadily over a period of at least eighteen months, even though the eye was apparently healed. For a few patients the dose was further increased, for one patient until the final dose of 500 mg. was reached, and this amount was given steadily at weekly intervals. For these patients the minimum period of treatment was two years, and treatment was then abandoned only if comparative nonreactivity of the skin had been attained and the condition of the eye was satisfactory. After treatment had been discontinued, the cutaneous reactivity was redetermined at the end of another three months. If the reaction was still negative, treatment was continued, but the cutaneous reactivity to tuberculin was determined regularly at three month intervals. With any return of cutaneous sensitivity, treatment with tuberculin was recommenced imme-

TABLE 3.—*Results of Tuberculin Therapy in Patients Treated Steadily Over a Minimum of Two Years*

Ocular Involvement	Number of Cases	Results		
		Healed	Improved	Unimproved
Keratitis..........................	2	2	0	0
Kerato-iritis....................	8	6	2	0
Scleritis.........................	0	0	0	0
Posterior ocular tuberculosis..	19	11	6	2
General uveitis................	3	3	0	0
Total......................	32	22	8	2
Percentage................	100	68.7	25	6.3

diately, again with a small dose at first as indicated by the degree of cutaneous reactivity. In several patients we have seen the recurrence of cutaneous sensitivity antedate the recurrence of activity in the eye. On this system the greater number of these patients have been followed for over five years, and in 4 patients with solitary tubercles of the choroid, tuberculous periphlebitis, tuberculous kerato-iritis and nodular iritis, respectively, the period of observation has now been over ten years. The results of treatment in this group of patients are shown in table 3. Twenty-two, or 68.7 per cent, appear totally healed. Eight, or 25 per cent, are definitely improved, with the disease under full control, but not as yet considered completely healed. Two patients showed no improvement. One of these patients had long drawn out generalized uveitis, which finally quieted but left a secondary cataract. The second patient's was distinctly an "uncompensated case," with severe, persistent scleritis and recurrent iritis. She was violently hypersensitive to tuberculin and showed repeated reactions to minute doses. She has never been able to take a larger dose than 1 mg. Over a period of six years, time and again we have been forced to abandon tuberculin therapy in this

case. This patient is included in the series only because from the first we purposed to treat her on the basis of the desensitization concept and despite repeated efforts have been unable to continue.

These two groups are not strictly comparable, owing to their difference in size, but they show a striking difference in the results obtained by the two methods of treatment. An obvious criticism is that the better results obtained by the treatment based on the desensitization concept are a reflection of a longer period of observation, that ocular tuberculosis tends naturally to healing and encapsulation and therefore the better results obtained in patients treated and observed over a minimum period of two years cannot fairly be attributed to the effect of tuberculin. In answer to this criticism we have clinical records for at least two years or over for 74 of the patients treated by one course of tuberculin, or under the perifocal concept. The results in this group are shown in table 4. Forty-two of these patients, or 56.7 per cent, still

TABLE 4.—*Results of Tuberculin Therapy with One Course of Treatment Only After Observation of Over Two Years*

Ocular Involvement	Number of Cases	Results		
		Healed	Improved	Unimproved
Keratitis..........................	11	8	2	1
Kerato-iritis.....................	12	5	4	3
Scleritis..........................	3	2	0	1
Choroiditis and retinitis.......	32	18	7	7
Uveitis...........................	17	9	2	5
Total......................	74	42	15	17
Percentage................	100	56.7	20.3	23

showed inactivity or healing of the original lesions; 15, or 20.3 per cent, continued to show improvement, while 17, or 23 per cent, showed no improvement, or had continuous relapses. These figures show no appreciable difference from the observation at the conclusion of the one course of tuberculin and indicate that the time element is not the factor responsible for the better results obtained in the group treated on the basis of the desensitization concept.

We have no figures of the results in a control series of patients with comparable ocular tuberculosis not treated with tuberculin. Hartig,[7] in his study of the effect of tuberculin treatment in ocular tuberculosis, withheld tuberculin for one year from a control group of 14 patients with this disease. In only 1 of these patients did he observe clinical improvement, while in the remaining 13 the disease remained unimproved, progressed or relapsed.

7. Hartig, F.: Ueber den gegenwärtigen Stand der Behandlung den Augentuberkulose, Ztschr. f. Augenh. 50:79, 1923.

Table 5 shows the results of our total experience with tuberculin therapy in the 175 patients studied, whether treated on the basis of the perifocal concept of action or that of the desensitization concept. Ninety-six, or 54.7 per cent, on last observation appeared healed; 52, or 29.7 per cent, showed improvement, while in 27, or 15.4 per cent, the condition was unimproved or the eyes had progressed to blindness.

General and Climatic Treatment.—In treatment based on the desensitization concept of the use of tuberculin the obvious aims are to remove, by the use of tuberculin, the fatal tissue hypersensitivity responsible for the inflammatory, destructive phases of the lesion and, by other means, to promote the acquired immunity which favors the encapsulation and destruction of the bacteria. At the present time, unfortunately, there is no means of artificially increasing the immunity which to a greater or lesser degree follows any tuberculous infection. An attempt to this end has been made by the vaccination of both children and

TABLE 5.—*Total Results of Tuberculin Therapy in Patients with Ocular Tuberculosis*

Ocular Involvement	Number of Cases	Results		
		Healed	Improved	Unimproved
Keratitis..........................	25	12	12	1
Kerato-iritis.....................	25	12	9	4
Scleritis..........................	7	5	0	2
Posterior ocular tuberculosis..	81	46	22	13
General uveitis.................	37	21	9	7
Total......................	175	96	52	27
Percentage...............	100	54.9	29.7	15.4

adults with BCG and killed virulent organisms.[8] At present, owing chiefly to the marked increase in the allergy which follows the injections, such vaccination is impossible in ocular tuberculosis. There are, therefore, only general means of promoting the acquired immunity.

The importance of rest, freedom from overstrain, cleanliness, fresh air, sunshine and an adequate diet in the treatment of any form of tuberculosis is recognized. For the patients reported on in this group, every effort has been made, so far as the social condition of the patients permitted, to meet these elementary aims. These simple hygienic regulations are of the greatest importance, and their proper maintenance, often with a change in environment, has apparently frequently marked the turning point in the course of the ocular disease. Nevertheless, in no case have we advocated sanatoriums or high altitude for the usual patient with ocular tuberculosis. The reasons for this are twofold. First, in the usual remote sanatorium for persons with tuberculosis it is impos-

8. Goodwin, T. C.: The Use of Protective Vaccines in Tuberculosis with Especial Reference to B.C.G., Internat. Clin. **2**:249, 1936.

sible for the patient to receive the careful observation of his ocular lesion necessary for successful tuberculin therapy. Second, in spite of Werdenberg's[9] contention that treatment with high altitude tends to lessen the dangers of untoward tuberculin reactions, we can see no reason why climatic treatment with high altitude should influence the course of ocular tuberculosis uncomplicated by active pulmonary lesions. Our experience in the coincidence of ocular tuberculosis with active pulmonary tuberculosis, which will be reported in detail elsewhere, does not agree with that of Werdenberg. While we do agree with the many investigators that ocular tuberculosis is invariably secondary to systemic tuberculosis and usually to infection of hilar glands, we have not been able to demonstrate constantly, either by physical or by roentgen ray examination, the pulmonary involvement invariably observed by Werdenberg. It appears either that we must be in error in our clinical diagnosis of ocular tuberculosis or that American radiologists must read as normal many of the 60 per cent of roentgenograms of the chest interpreted by Werdenberg as showing slight but definite evidences of tuberculosis of hilar glands or the lungs. In regard to treatment in sanatoriums and with high altitude, it is of some interest that 3 patients in this series came directly to Baltimore from sanatoriums for persons with tuberculosis where their ocular tuberculosis had progressed unfavorably. Two of these patients are now classed as healed, and 1 is classed as improved.

SPECIAL PROCEDURES

Paracentesis and Autohemotherapy.—These procedures are indicated especially for patients with the so-called "uncompensated" types of ocular tuberculosis, patients for whom Werdenberg[9] believes tuberculin is contraindicated. These patients usually have tuberculosis of the anterior part of the uvea, are extremely hypersensitive to tuberculin and show a rather intense, long-continuing inflammatory reaction, with a tendency to extension rather than limitation. They usually take tuberculin poorly, with local and sometimes focal reactions. Theoretically, in these patients one would imagine an excess of antigen, or tuberculoprotein, with spreading sensitization of the ocular tissues and an insufficient concentration of immune bodies in the ocular humors. These patients usually show an amazing improvement in the clinical picture, and may become "compensated" after paracentesis of the anterior chamber and drainage of the aqueous, a procedure which theoretically is accompanied by a large increase in immune antibodies in the reformed aqueous. We do not agree with Werdenberg that tuberculin is absolutely contraindicated in this group of patients, but we do agree that

9. Werdenberg, E.: Beurteilung und Behandlung der Augentuberkulose. Klin. Monatsbl. f. Augenh. (supp.) 94:3, 1935.

it must be used in extremely small doses and with great caution until the intensity and extension of inflammation have been controlled by such a procedure as drainage of the aqueous or until the lesion has become compensated.

Autohemotherapy, or injection of the patient's own blood into the anterior chamber, the procedure advocated strongly by Schieck,[10] has, in our opinion, little advantage over simple drainage of the aqueous. The further concentration of antibodies incidental to the injection of such a small amount of whole blood into the anterior chamber must be slight. The beneficial effects reported for autohemotherapy may all fairly be attributed to the drainage of the aqueous incidental to this procedure. Our experience with simple drainage in these cases has been equally as satisfactory as when this was used with the added step of autohemotherapy, which always carries with it a slight additional risk of infection.

Phototherapy.—A. Ultraviolet Rays: Both general and local irradiation with ultraviolet rays have been used in the treatment of ocular tuberculosis. Duke-Elder[11] stated the belief that the effect of general irradiation of the body is due to complex photochemical and photoelectric changes, while the local effect is abiotic, with photochemical denaturation and coagulation of the protein. Duke-Elder,[11] Goulden,[12] Koeppe[13] and others have reported strikingly beneficial results in tuberculous iridocyclitis by general irradiation of the body, and in a few cases apparently good results with local irradiation of the diseased eye. On the other hand, Birch-Hirshfeld and Hoffman[14] expressed the opinion that ultraviolet irradiation is only an adjunct in the treatment of ocular tuberculosis.

We have used ultraviolet irradiation in a number of cases of tuberculosis of the anterior ocular segment. General irradiation with the use of a quartz mercury vapor lamp with doses just below or at the skin erythema dose produces a sense of well-being, improves the general tone of the patient and certainly does not aggravate the ocular lesion. On the other hand, we have observed no marked beneficial results

10. Schieck, F.: Die Behandlung von tuberkulösen Erkrankungen des vorderen Bulbusabschnitts durch Eigenblutinjektionen in die Vorderkammer, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **49**:183, 1932.

11. Duke-Elder, W. S.: Ultra-Violet Light in the Treatment of Ophthalmic Disease: I. General Phototherapy, Brit. J. Ophth. **12**:289, 1928; II. Local Phototherapy, ibid. **12**:353, 1928.

12. Goulden, C.: The Value of Ultra-Violet Rays, Tr. Ophth. Soc. U. Kingdom **46**:207, 1926.

13. Koeppe, L.: Die Diathermie und Lichtbehandlung des Auges, Leipzig, F. C. W. Vogel, 1919.

14. Birch-Hirschfeld, A., and Hoffman, W.: Die Lichtbehandlung in der Augenheilkunde, Berlin, Urban & Schwarzenberg, 1928.

which could reasonably be attributed to such treatment. Local irradiation with the use of the Birsch-Hirshfeld carbon lamp, with exposures of from thirty seconds to one minute, have been used almost as a routine in the treatment of deep scleritis and in some cases of kerato-iritis, due care being taken to protect the lens. In deep scleritis our general impression has been that the treatment has been of some value, but in no case have the results been striking. Local ultraviolet irradiation appeared to have little if any effect on corneal infiltrates or scars and did not appear to influence the course of iritis.

B. Roentgen Ray Treatment: The use of roentgen ray irradiation in ocular tuberculosis has been advocated by the German ophthalmologists, notably Stock,[15] Scheerer[16] and Werdenberg.[9] The most favorable results have been reported in nodular iritis, scleritis and keratitis. Stock reported poor results in cyclitis and choroiditis and no results in retinal periphlebitis. He reported the optimum dose to be 20 per cent of the skin erythema dose, and Scheerer, after first using a larger dose, now advocates a dose between 10 and 15 per cent of the skin erythema dose. Within from twelve to twenty-four hours after the irradiation of the eyes there ensues a more or less violent irritation, which disappears within three to four weeks, with marked improvement or even healing of the lesion. If necessary, the treatment may be repeated until a total of about 80 per cent of the skin erythema dose has been given. Scheerer recommends the use of a 0.5 mm. zinc and a 3 mm. brass filter, with a field measuring 4 by 4 cm. He believes the beneficial effects are due to degeneration of the lymphocytes and leukocytes in the lesion, while Werdenberg believes the good effects are due to the roentgen rays producing perifocal reactions about the lesion similar to those produced by tuberculin.

We have used roentgen ray therapy in only a few cases, and our experience has not been so fortunate as that just reported. The technic employed has been slightly different, but the dose is about the same. A 1 mm. aluminum filter and a 2 by 2 cm. field were used, and a calculated dose of 140 roentgens was given. Since 800 r. is about the skin erythema dose, this dose was apparently 17 per cent of the skin erythema dose, midway between the 15 per cent advocated by Scheerer and the 20 per cent recommended by Stock. We observed the same activation of the lesion reported by these authors but failed to observe cicatrization or healing of the lesion after the more or less violent primary reaction subsided. In fact, the primary activation appeared to aggravate the

15. Stock, W.: Strahlenbehandlung in der Augenheilkunde, Klin. Monatsbl. f. Augenh. **76**:542, 1926.

16. Scheerer, R.: Röntgenbestrahlung bei Uvealtuberkulose, Klin. Monatsbl. f. Augenh. **75**:27, 1925.

progress of the lesion, and we have abandoned roentgen ray treatment. Possibly, had a greater number of patients been treated or the treatment persevered in, the beneficial effects reported by others might have been observed. As it is, our limited experience has frankly been discouraging.

C. Radiotherapy: The use of radium for ophthalmic conditions and ocular tuberculosis has been studied, especially by Kumer and Sallman,[17] who have published a comprehensive monograph on the subject. They recommend radiotherapy, especially in tuberculous keratitis and iridocyclitis. In keratitis they note specifically the theoretical advantages of using the only slightly penetrating soft beta rays. However, since there are very few beta ray applicators in the world, they have used chiefly the gamma rays and a small amount of the hard beta rays. They used 100 mg. of the element in a 0.3 mm. platinum carrier which transmits 97 per cent of the gamma rays and 6 per cent of the hard beta rays (Fernau). In keratitis they have recommended direct or almost direct contact with the cornea and less than the skin erythema dose. In cases in which the condition is acute they used from 20 to 30 per cent of the skin erythema dose at intervals of two weeks. In tuberculous iridocyclitis they have employed either from 50 to 60 per cent of the skin erythema dose at one application or from 30 to 50 per cent of the skin erythema dose repeated at two week intervals and given at a distance from the eye. In general they reported good results in tuberculosis of the anterior ocular segment but noted that several patients were not helped and that in a few in whom the condition was active it was seriously aggravated. No effect was noted in juvenile retinal periphlebitis.

Our first experience with radiotherapy in ocular tuberculosis was confined to irradiation with the gamma rays, the dose being 1 gram minute at $\frac{1}{16}$ inch (0.2 cm.), approximately 50 per cent of the skin erythema dose. Only patients with kerato-iritis were treated. The results were unfortunate, although in no case was any permanent harm done. Definite activation of the uveal inflammation came on shortly after the treatment, persisted for some weeks and was not followed by a compensatory secondary phase of healing. It is true that there did appear to be some clearing of the corneal scars. Recently, through the agency of Dr. Curtis F. Burnam and the Howard A. Kelly Hospital, Baltimore, there has been available for use a beta ray applicator. The energy usually employed has been approximately 500 mg. of radon emanation in only the thinnest possible glass container, without a filter. This filter turns back any alpha rays and transmits practically all the soft beta rays and the gamma rays. The applicator is used in

17. Kumer, L., and Sallman, L.: Die Radiumbehandlung in der Augenheilkunde, Berlin, Julius Springer, 1929, p. 198.

direct or nearly direct contact for five seconds, the dose therefore being 2.5 gram seconds. This is repeated at two or three week intervals. This is approximately 50 per cent of the skin erythema dose of the beta rays and only about 4 per cent of the skin erythema dose of the gamma rays. The soft beta rays penetrate only from 2 to 3 mm. In the few patients treated to date we have observed not the slightest reaction and no activation of the uveal lesion. The results on the corneal infiltrates and scars have been remarkably good. There has resulted definite clearing of the cornea, and in one patient, who had a clear lens and a normal fundus, vision has improved from 20/100 to 20/50. Beta ray irradiation appears free from danger to the iris and lens and offers distinct promise in the treatment of corneal tuberculosis and in the clearing of corneal scars.

Subconjunctival Injections.—Subconjunctival injections of physiologic and hypertonic solution of sodium chloride have been used in a few patients with indolent inflammatory lesions in whom there was apparently little tendency to absorption and some mechanical procedure to hasten the process appeared in order. When daily subconjunctival injections of physiologic solution of sodium chloride were tolerated without reaction, the strength of the solution was gradually increased to a maximum of 3 per cent. These injections, with the attendant diffusion of fluids produced by different concentrations of sodium chloride beneath the conjunctiva and within the eye, appeared to promote the clearing of exudates and marginal corneal infiltration. Certainly no marked or dramatic change for the better followed their use, but there was likewise no activation of the lesion. While we are not unduly enthusiastic about the results obtained by subconjunctival injections of hypertonic solution of sodium chloride, on the whole they appear to be of some value in hastening absorption in cases in which the lesion is indolent.

<div align="center">COMMENT</div>

In our early experience with tuberculin, influenced by the perifocal concept, the custom was to discontinue tuberculin when the dose of 9 cc. of no. 2 solution, or 90 mg., was reached. If the patient later showed a recurrence of ocular inflammation, tuberculin therapy was recommenced, the degree of cutaneous reactivity being again ascertained in order to determine the proper initial dose of tuberculin. At this time it was noted that the recurrence of the ocular inflammation was usually accompanied by a marked degree of cutaneous reactivity. It was also noted that a few patients under tuberculin therapy showed sudden changes and increases in the cutaneous sensitivity, which necessitated decreasing the therapeutic dose. This change in sensitivity often preceded a flare-up or recurrence of the ocular inflammation. In order to

ascertain if there was any relation between the apparent return or increase in the hypersensitivity to tuberculin and the recurrence of activity of ocular inflammation, the degree of cutaneous reactivity was determined at the end of a course of tuberculin therapy and redetermined at three month intervals thereafter. It was found that after tuberculin was discontinued there was often a steady return of the cutaneous reactivity, and this often preceded and apparently foretold the recurrence of ocular inflammation. In the light of this observation, tuberculin treatment was recommended on a return of the cutaneous sensitivity and before the expected ocular recurrence materialized. This procedure was sometimes unsuccessful, the ocular relapse occurring despite the treatment. We believed these recurrences could be explained on the grounds that there had been insufficient time to achieve any appreciable amount of ocular desensitization. We therefore approached the problem on the basis of the desensitization concept, the theory that tuberculin should be given with the idea of achieving tissue desensitization and must be steadily continued in order that this desensitization might be maintained and, as far as possible, made permanent. Our present ideas are that tuberculin must be given for a minimum of two years and discontinued then only if the skin has become almost totally nonreactive, and the ocular lesion has been entirely quiet and apparently encapsulated for a period of a year or longer. Even in such cases as these, whenever possible we redetermine the cutaneous sensitivity at three month periods and begin tuberculin therapy again with any sign of return of the cutaneous sensitivity, irrespective of the entire quiescence of the ocular lesion.

Occasionally patients are encountered who appear incapable of being desensitized. These patients are exquisitely sensitive to tuberculin and constantly show local, apparent systemic, and even focal, reactions to infinitesimal doses of tuberculin. Such patients may belong to the uncompensated type described by Werdenberg and be similar to one of the patients reported on in our series treated on the basis of the desensitization concept in whom treatment failed or may be patients with recurrent, brief inflammatory flare-ups in the eye, which are apparently related to the parenteral absorption of any amount of tuberculin. We have seen at least 2 patients of the last class. The only possible method of treating these patients with tuberculin would be to begin with a dose of 0.000001 mg., or even less, below their point of reactivity, whatever that may be, and increase the dose only with the patient under rigid observation.

The question of the influence of tuberculin treatment on recurrences of the ocular inflammation is difficult to analyze. We have lost track of almost half of the 143 patients treated on the basis of the perifocal

concept and therefore have no accurate figures for this group. Of the patients we have been able to follow, 36 are recorded as having shown one or more relapses, a total of 67 recurrences being noted. The majority of these recurrences were severe. The 32 patients treated on the basis of the desensitization concept, who have received tuberculin for a minimum of two years, have been more carefully followed. Twelve of these patients have shown recurrences. Ten have had one recurrence only, 1 had three minor recurrences and 1 a number of minor recurrences. With one exception, all the recurrences in this group were of low intensity and occurred rather early in the course of treatment, well before either systemic or ocular desensitization could have been accomplished. The severe recurrence occurred after four years of steady treatment. This recurrence appears to have been precipitated by the persistent injection of an excessive amount of tuberculin, to which the patient was definitely showing local cutaneous reactions. Happily, this patient recovered without residual damage after repeated paracentesis and the proper adjustment of the dosage of tuberculin. It is our distinct impression that the recurrences have been fewer and of less intensity in the patients treated on the basis of the desensitization concept than in the patients receiving only one course of tuberculin. Certainly we have seen no lost, phthisical eyes in the patients given prolonged tuberculin therapy, while such eyes have been observed in the group of patients who received what we now believe to have been inadequate treatment.

SUMMARY AND CONCLUSIONS

The results of tuberculin therapy and certain special forms of therapy in 175 patients with conditions diagnosed as various types of ocular tuberculosis are reported. One hundred and forty-three of these patients received only one course of tuberculin, being treated on the basis of what may be termed the perifocal concept of the action of tuberculin— the theory that tuberculin acts by causing small, subacute reactions about the ocular lesion, thus producing local immunity. Thirty-two patients received tuberculin more or less constantly over a minimum period of two years, being treated on the basis of what may be termed the "desensitization" concept—the theory that tuberculin acts by actually desensitizing the ocular tissues and so removing the factor responsible for the inflammatory and destructive phases of the tuberculous lesion. The outcome in the patients treated on the basis of the desensitization concept was decidedly better, both as to end-results and recurrences, than the results obtained in patients treated on the basis of the perifocal concept. From the theoretical, experimental and practical points of view, we believe the desensitization concept for the action and use of tuberculin

is correct and that tuberculin should be used in the treatment of ocular tuberculosis with the end-view of achieving and maintaining tissue desensitization.

General hygienic treatment is always indicated in ocular tuberculosis. Special procedures such as paracentesis, autohemotherapy, ultraviolet irradiation and subconjunctival injections are indicated in certain groups of patients. Our experience with roentgen ray therapy and gamma ray therapy has not been encouraging. Very encouraging results have been observed in our limited experience with beta ray radium therapy.

CORTICAL INNERVATION OF OCULAR MOVEMENTS IN THE HORIZONTAL PLANE

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A physiologist to whom ophthalmologists owe some of the best devised experiments on the mechanism of oculomotion recently expressed his opinion on the cortical innervation of ocular movement. This opinion, as expressed by him in various publications, is essentially in agreement with that of Duval and Laborde, although the latest anatomic researches show such a view to be untenable. Spiegel and Scala,[1] while apparently admitting the correctness of the latest data, viz., that the nucleus commissurae posterioris plays an important part in the mechanism of horizontal ocular movement, as does also a lateral nucleus in the globus pallidus as a tertiary center, stated: "There still exists much uncertainty concerning the cortical innervation of the ocular muscles." There seems to me to be some discrepancy in the reasoning of Spiegel and Scala, for, while admitting the uncertainty of the cortical innervation of the ocular muscles, they nevertheless have taken for granted, as shown by a full consideration of the results of their experiments, the existence of cortical centers for oculomotion and, further, that these stimuli are conducted by the capsula interna. As such controversy is a serious obstacle to progress in this field, I consider that the article written by Spiegel and Scala in clear and plausible style should not remain unanswered by those of the opposite camp.

As I have worked many years along purely physiologic lines (in the laboratories of Engelmann and Bowditch), Spiegel's close association with the physiologic side of the question and my own present association with anatomiconeurologic research need not exclude the possibility of our arrival at our common goal, i. e., a clear understanding of this most mysterious and evasive problem, the innervation of coordinated ocular movements.

This problem seems to me to appeal to the physiologist, the anatomist and the clinician, particularly ophthalmologists, otologists and neurologists, whose work brings them daily into contact with the practical side of the problem. At the same time it must be realized that any theory put forward by any one of these groups will be instantly criticized by the others. My own approach to the problem is along anatomico-

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† Dr. Muskens died June 11, 1937.

1. Spiegel, E., and Scala, N. P.: The Cortical Innervation of Ocular Movements, Arch. Ophth. **16**:967 (Dec.) 1936.

physiologic lines in a research on forced movements. Since the mechanism operative in these forced movements in their three planes is the same as for ocular movements in their three planes (as shown in my recent book [2]), I am entitled to state: The permanent forced horizontal position, for instance, to the right, as seen in the human object and appearing in the experimental animal as horizontal conjugated deviation (with circular movement) to the right, occurs after cross-section of the left tractus vestibulomesencephalicus cruciatus, as well as after lesion of the right nucleus commissurae posterioris, the right nucleus lateralis globi pallidi and the connecting fiber tracts between the two latter structures. No permanent forced movement or position results in the human subject, or in the experimental animal from a lesion of any other part of the central nervous system. It is particularly to be observed that this movement never follows lesions limited (by anatomic control) to the cortex cerebri or to the pyramidal tract, no matter how extensive these lesions are.[3]

Is one therefore in a position to deny the existence of any cortical influence on coordinated ocular movements? It must be admitted that one cannot go so far as this, but at least one can approach the question with an open mind, if somewhat cautiously. There can be no doubt that direct nervous connections exist between the pallidum and other structures, i. e., the neostriatum, the thalamus and the mesencephalon. Stimulation of the latter structures may induce ocular movements. As no direct fiber connection between the cortex and the pallidum is known, such stimuli must travel along an indirect path, i. e., the cortex, the thalamus, the pallidum, the nucleus commissurae posterioris and the tractus commissuromedullaris, thus demonstrating that ocular movements may result from stimulation of the cortex.

Such a question requires careful investigation and the task has been made much easier by such workers as D. Spiegel, who has elaborated a skilful method of examining vertical movements. It is interesting to remember that this very point baffled such an eminent worker as R. Russell, even with Hughlings Jackson as his advisor! [4]

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2. Muskens, L. J. J.: Das supravestibuläre System, Amsterdam, N. V. Noord-Hollandsche Uitgevers Maatschappij, 1934, chaps. 2, 9, 11 and 16.

3. Muskens,[2] chap. 22.

4. Spiegel and Teschler's conclusion (Arch. f. d. ges. Physiol. **222**:370, 1929) that the vestibular nuclei are a station by which cortico-oculomotor innervation has to pass seems to me not warranted. Not only do anatomists know of no fiber relation between the pyramidal tract and the vestibular nuclei, but another view seems more acceptable. According to the modern notions, the vestibular nuclei, with their ascending—and, from the commissural nuclei, descending—connections produce the necessary background on which striatal, tectal or cortical (by some roundabout way) innervation has to play. This well attuned background lacking no properly coordinated horizontal ocular movements can occur.

When one recalls the work of Luciani and Tamburini, who regarded ocular movements following cortical faradization as being dependent on escape of current through the brain tissue [5] and, further, that clinicians and anatomists profess complete ignorance of any cortical tracts which convey oculomotor stimuli, it is obvious that this is a matter about which one must be very doubtful. That the secondary and tertiary supravestibular nuclei, which certainly play a part in the production of coordinated horizontal ocular movements, may receive stimuli in some more or less complicated way via collateral fiber tracts from neostriatal and cortical structures (Monakow) no one would deny.

But the point at issue is: Can it be proved beyond doubt that there is a direct fiber tract from some cortical area by way of the pyramidal tract and the capsula interna which, either on cross-section or on stimulation, disturbs or induces coordinated ocular movements? In my opinion, this cannot be proved, and on this point I differ from Spiegel and Riley, who, with others, have affirmed unhesitatingly that there is such a tract.

How did the idea that the pyramidal tract is associated with oculomotion innervation originate? Starting from the assumption that all voluntary movements are innervated from the pyramidal tract, Dejerine submitted the possibility that oculomotion follows the usual rule of voluntary innervation. Windler suggested that the aberrant pyramidal tract may be responsible, while Spiller discussed the matter in the same light but in more detail. In this way the idea took firm root, although proof was lacking, and the fact that the oculomotor mechanism exists in lower animals in which the pyramidal tract is not yet elaborated was overlooked, as was also the fact that oculomotion in three planes in those lower animals was part and parcel of the general body locomotion. At the same time, it must be pointed out that Leyton and Sherrington found that the mechanism of oculomotion is differently organized from that of voluntary muscular movements, while Wernicke considered ocular movements to be probably reflex, the impulse alone being voluntary.[6]

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5. If one compares the method used by Ken Taga (Arch. f. d. ges. Physiol. **223**:117, 1929), who got better results when he stimulated a locality nearer the pallidum, viz., the capsula interna, with the old experiments of François Franck, it appears as if the Italian authors hit not far beside the mark. When judging the influence of the cross-section of the posterior commissure on the coordinated ocular movements it is well to recall that according to Godlowski's stimulation experiments Ganser's ventral commissure might compensate for the loss of function of the dorsal (i. e., posterior) commissure.

6. In regard to the reflex gaze, which is often present in cases of horizontal conjugate deviation of the head and eyes, caused by destruction of the globus pallidus (Muskens, L. J. J.: Das supravestibuläre System, Amsterdam, N. V. Noord-Hollandische Uitgevers Maatschappij, 1934, chap. 16) and its experimental

The clinician is well aware that in case of hemiplegia in which the capsula interna is completely destroyed ocular motion is rarely affected unless the striatum (more accurately, the globus pallidus) is involved (Prévost). And in a case of conjugate deviation with persistence of the simple vestibular reflexes, one is entitled to diagnose an intact fasciculus longitudinalis posterior and a lesion of the pallidum or of its connection with the commissural nuclei. Further, in experiment, as in disease, nystagmus, with a slow jerk to the right, is the first step toward conjugate deviation to the right,[7] and the notion had to be given up that the quick jerk of supravestibular nystagmus originates in the cortex (Bauer and Leifler, de Kleyn, Wilson and Pike, and Ivy). All this will be found in full accordance with the modern notions.

There is at least enough basis for the argument that the oculomotor mechanism ought to be regarded as a phenomenon sui generis. Without regard to other (at least pyramidal tract) motor mechanisms, I submit that the proper method of approaching the question of the mechanism of oculomotion is consideration of anatomicophysiologic data (two fundamental experiments are described in my work[8]) which are based on the (controlling) experience of twenty-three years of physiologic, anatomic and clinical work, which experience serves excellently as a foundation on which a theory or doctrine regarding the innervation of coordinated oculomotion could be built.

If in a larger number of lesions of the brain stem involving division of tracts or disturbance of nodal points up to the pallidum but not farther the coordinated movement is found to be disturbed, is it not better to keep firmly to known physiologic facts rather than to sail away before the capricious winds of fantasy?

Further, should it not be remembered that such authors as Leidler[9] the otologist, Nordmann[10] the ophthalmologist and Godlowski[11] the

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corollary, Graham Brown's orientation reflex (Arch. néerl. de physiol. 7:344, 1922), there can be little doubt left about its localization. For Graham Brown found the reflex present as long as the posterior commissure and the fasciculus longitudinalis posterior was intact, and absent after cross-section through the corpora quadrigemina posteriora. As far as I can see, this result is in entire concordance with the new doctrine, according to which the infracommissural nuclei are the nodal points for reflex locomotion and oculomotion.

7. For this reason it seems now to be generally admitted that the slow jerk is the main feature of the syndrome, the past pointing being directed in the same sense. Hence I urged—till now in vain—that the direction of the nystagmus should be named after the direction of the slow jerk.

8. Muskens,[2] chap. 9.

9. Leidler, R.: Fragen der Lokalisation innerhalb des zentralen vestibular Systems, Monatschr. f. Ohrenh. 70:267, 472, 544, 725, 801 and 951, 1936.

10. Nordmann: Personal communication to the author.

11. Godlowski: Podkowa osradki ipojzzenia I, Krakow, 1936.

neurologist have admitted that these novel views are far preferable to the antiquated ones already referred to? Of course, the outlook would be different if the two fundamental experiments, the bedrock from which I started, were proved to allow of other interpretation. This is the weakest link in the chain, and if anatomists and physiologists like Spiegel and his colleagues would either confirm these comparatively simple experiments (in which osmium-stained control preparations were used) or demonstrate a fallacy in them, the first step toward the final solution of this irritating riddle of oculomotion would be made.

DETACHMENT OF THE RETINA

OPERATIVE RESULTS IN ONE HUNDRED AND SIXTY-FOUR CASES

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AND

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In the 164 cases of retinal detachment analyzed in this paper the eyes were operated on in the Institute of Ophthalmology of the Presbyterian Hospital, New York, between April 1, 1934, and Jan. 1, 1937. The total number of eyes operated on was 171. No attempt was made to select the cases, as it is still our feeling that almost all eyes in which every possibility of spontaneous cure has been eliminated should be offered a chance of benefit by surgical intervention. This attitude has led us to attempt treatment in many so-called hopeless cases. The results in this type of case were, on the whole, disappointing, yet surprising recoveries occurred sufficiently often to renew our interest in these very unfavorable cases. Some form of diathermy was used in practically all the cases, with an occasional minor variation in operative technic.

GENERAL STATISTICS

Sex.—In this series of 164 patients 67 per cent were males and 33 per cent were females. This difference is at least partially accounted for by the higher incidence of frank trauma in the males. A definite history of trauma was obtained in 21 per cent of the males and in only 9 per cent of the females.

Age.—The youngest patient was 6 years of age, and the oldest 79 years. In each decade between the ages of 20 and 60 there were but slight differences in the number of cases. There were 7 patients under 10 years of age in this group. Four of the adults were more than 70 years old.

Trauma.—In this series of 171 eyes a positive history of trauma was obtained in 29 cases (17 per cent). We were careful to include in this group only cases in which there was a definite causal relationship between the injury and the onset of the detachment. In 23 other cases (13 per cent) the accident was considered to be at least a contributory

factor. A history of trauma was obtained from only 10 per cent of the patients with high myopia, while 23 per cent of those with low refractive errors (exclusive of aphakic patients) gave a history of injury. A cure was effected in 62 per cent of the cases in which trauma was definitely established as the causative factor.

OCULAR FINDINGS

In the cases of unilateral retinal detachment no preference was found as to the eye involved. If this series is combined with the one we [1] reported in 1934, there are 314 cases. In this group the right eye alone was affected 131 times and the left eye alone 128 times. Both eyes were involved in 55 instances, thus making the detachment bilateral in 17.8 per cent of all the cases. The frequency with which the fellow eye sooner or later becomes involved is striking, and in all probability if this series is followed for a longer time this percentage will rise. This fact should serve as an added stimulus to ophthalmologists to conserve all possible vision in every case of retinal detachment. Analysis of the 31 cases of bilateral retinal detachment in the present series showed 15 of the patients to have high myopia (requiring a sphere of over —6.00 D.) and 7 to be aphakic.

Refraction.—Analysis of the refractive error in this series of patients revealed myopia to be present in 53.8 per cent. In the series reported on in 1934, 66.6 per cent were myopes. These figures, along with those reported by others show that some degree of myopia exists in at least 50 per cent of all cases of retinal detachment. It is interesting to note that myopia of 6 D. or over was present in 30.4 per cent of this group. Of the 13 aphakic patients in this series, 5 had high myopia.

Muscular Imbalance.—In the present series no annoying postoperative diplopia resulted from the severance and reattachment of the rectus muscles. In a few instances a small amount of deviation was detected on routine postoperative tests of muscles, which, however, was usually insufficient to require a prismatic correction. This fact clearly indicates that one need have no fear about detaching one or more of the rectus muscles whenever such a procedure is necessary for a good operative exposure.

Intra-Ocular Tension.—As was suggested in 1934, hypotony is a grave prognostic sign. In the present series there were 17 instances in which the preoperative intra-ocular tension was below 10 mm. of mercury (Schiötz). Operations in these 17 cases resulted in 15 failures.

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1. Dunnington, J. H., and Macnie, J. P.: Detachment of the Retina: Operative Results in One Hundred and Fifty Cases, Arch. Ophth. **13**:191-200 (Feb.) 1935.

It was noted in our previous article that in 5 instances in which the preoperative intra-ocular tension was less than 6 mm. of mercury no cures were effected. Hypotony is usually found along with other unfavorable signs, e. g., extensive detachment, but it is occasionally encountered in a person with detachment who appears in all other respects to be a good operative risk. It is therefore sugggested that the intra-ocular tension be recorded preoperatively in every case of retinal detachment.

Increased intra-ocular tension occasionally accompanies a serous detachment of the retina, as was noted in 6 of our cases. In no instance was there a return of the glaucoma after the operation for the retinal detachment. The retina was satisfactorily reattached in 3 of these 6 cases.

Changes in the Crystalline Lens.—Complete opacification of the lens developed postoperatively in 7 of the cases in which the results of operation were unsuccessful. Lenticular opacities were frequently found preoperatively in the patients over 50 years of age, yet an increase occurred postoperatively in only 1 instance in which satisfactory reattachment was obtained. The rarity of a postoperative increase in the lenticular changes seems to indicate that the operation itself has no appreciable effect on the status of the crystalline lens.

Changes in the Field of Vision.—In all cases careful perimetric examinations were made before operation. The changes in the field were found to correspond closely with the area of the detachment. After operation, in certain cases a defect in the field persisted which seemed to correspond closely to the areas of damage to the retina and choroid produced by the operation.

In those cases in which the detachment involved the macular region, replacement of the retina was not followed by a complete return of central vision.

From an analysis of these cases we still feel as we did in 1934 that if careful perimetric tests are done almost all eyes will show some impairment of function. Furthermore, it is our opinion that the degree of reaction necessary for cure in itself produces some reduction in the sensitivity of the retina.

RELATION OF VARIOUS FACTORS TO THE OPERATIVE RESULT

Time of Onset.—The series was studied to determine the relationship between the time of onset and the operative result. When those cases in which approximately one half of the retina was detached were considered it was found that the duration of the detachment had little, if any, influence on the result. In this group cure was effected in approximately two thirds of those patients operated on within one month after the onset. In similar conditions of over three months'

duration cure was also effected in two thirds of the cases. Attention should be called to the difficulty in determining the exact date of the occurrence of the detachment. Only those cases were included in this study in which this date could be established with reasonable certainty.

Among the patients with approximately one half of the retina detached there were 7 in whom the detachment had existed for at least a year. In 4 of these cases a cure resulted after operation; the longest interval between the onset of the detachment and a successful operative result was four years. In the group with more extensive detachment of the retina (three fourths or more of the retina) a favorable result (cure or improvement) was obtained in approximately one fourth of those patients operated on within one month after the onset. The same percentage of cures was obtained in the cases of extensive detachment of over three months' duration.

Site of Detachment.—In those patients with approximately one half of the retina detached the inferior portion was affected in 50 per cent. Treatment confined to this region was successful in 74 per cent.

TABLE 1.—*Relation of Extent of Detachment to the Operative Result*

Ninety-Nine Eyes with One Half or Less of the Retina Detached		Seventy-Two Eyes with Three Quarters or More of the Retina Detached	
Result	Percentage	Result	Percentage
Cure	64.6	Cure	11.1
Improvement	4.1	Improvement	13.9
Failure	31.3	Failure	75.0

The temporal half was detached in 31 per cent, with involvement of the nasal or the superior parts in approximately 9 per cent. In these cases a successful result was obtained in between 55 per cent and 60 per cent. The figures just given illustrate that the location of the detachment has but little bearing on the outcome.

Extent of Detachment.—In this analysis an attempt was made to determine what rôle the extent of the detachment played in the operative result. This is strikingly shown in table 1.

The percentage of cures in those cases of detachment involving one half or less of the retina is most encouraging, but equally heartening is the fact that 25 per cent of the patients with extensive detachment can be benefited by operation.

Type of Detachment.—In this series the detachment was considered to be bullous in 65 per cent of the cases, mixed in 17 per cent and flat in 17 per cent.

Of those patients with a bullous detachment involving not more than one half of the retina, approximately 65 per cent were benefited by

operation. Among 18 instances in which one half or less of the retina was very slightly elevated, i. e., flatly detached, a cure was obtained in 14 (77.7 per cent).

The term mixed was used for those extensive detachments in which a bullous separation was accompanied by a slight elevation of a different portion of the retina. Consequently in this group the operative results were more unfavorable, failure occurring in 57 per cent of the cases.

Holes.—An attempt was made to seal all existing holes by concentrating the treatment in these areas, but, as was advocated in 1934, the entire area of the detachment was treated whenever feasible. The thought back of such widespread treatment is the desire to create an extensive area of adhesive choroiditis, which we feel insures a more permanent result. The consensus seems to be that the so-called "blind" operations are not often successful. However, a study of these cases shows that 54 per cent of the patients in whom no retinal holes were located preoperatively were benefited, while of those in whom one or more retinal holes were found before operation 48 per cent were either cured or improved. Analysis of the cases in which large or multiple holes or extensive disinsertions were found shows that a favorable result was obtained in 46 per cent. Among patients in whom only a single small hole was seen preoperatively a satisfactory result was obtained in 60 per cent.

DESCRIPTION OF OPERATIVE METHOD

Anesthesia induced by tribromethanol in amylene hydrate supplemented with ether was used during the majority of the operations. It is felt that there was little difference in the postoperative course between general and local anesthesia.

In all the operative methods the sclera was exposed over the extent of the detachment, and in most of the operations one or two of the rectus muscles were severed. In a few instances satisfactory exposure was obtained by retracting the muscles without detaching them.

In the majority of the operations diathermy was used. In approximately the first half of the period covered by this report (April 1, 1934, to Jan. 1, 1937) the Walker type of platinum needles was used with the Walker instrument. These were inserted into the sclera over the area corresponding to that of the detachment. The current was adjusted so that penetration took place with little pressure within a second or two after the contact was made. After all the needles were inserted they were withdrawn, the subretinal fluid being allowed to escape.

In the summer of 1935 a modification by Unsworth and Larkin of the Lacarrère electrodiafaco instrument was adopted in place of the platinum needles. With this method a wire of adjustable length can be used, permitting either surface application or penetration. The entire surface of the sclera corresponding to the area of the detachment was treated. From thirty to fifty applications were used, depending on the extent of the detachment. In some instances all the applications were perforating, while in others treatment was confined to surface coagulation except for a few final punctures to permit escape of the subretinal fluid.

Multiple trephining with chemical cauterization of the choroid (the method of Guist and Lindner) was used in but 10 instances in the early part of this series. This method has been discarded except when used in combination with one of the methods of diathermy, at which time one or more trephine openings are made, treated chemically and opened with a punctum dilator to insure better drainage of the subretinal fluid. The combined method was used in 49 operations.

Dr. Clifford Walker treated one patient with diathermy and injection of a 3 per cent solution of sodium hydroxide into the region of the macula and another by electrolysis. In each case cure resulted. One patient was operated on with the thermophore heated to 170 F. and applied for one minute to eight points in the inferior third of the globe between 12 and 17 mm. posterior to the limbus. One trephine opening 14 mm. posterior to the limbus below was made for drainage. This patient was cured by operation.

Postoperative care consisted in confinement to bed for approximately two weeks. During this time both eyes were bandaged and dressed every forty-eight hours, with the instillation of a 1 per cent solution of atropine sulfate into the affected eye. A gradual increase in activity was then allowed, and the patient was discharged from the hospital in about three weeks.

ANALYSIS OF RESULTS

In the entire group of 171 eyes operated on the detachment was either cured or improved in 50.3 per cent (cured in 42.1 per cent and improved in 8.2 per cent), while in 49.7 per cent failure resulted. The eyes were considered cured when the retina was completely reattached and there was enlargement of the field of vision. The "improved" eyes were those in which the field of vision became enlarged yet some peripheral detachment persisted or else the retina was in place but the field of vision was not materially enlarged.

In this series 23 patients had more than 1 operation. In 2 instances 4 operations were done. In one of these cases complete reattachment was eventually secured, while in the other the operations were unsuccessful. Of the 4 patients who had 3 operations, only 1 was benefited (improved). Twenty-seven eyes were operated on twice. Of these, 9 were cured and 5 improved.

An analysis of the 72 cases in which the eyes were cured showed that only 2 had a reduction in visual acuity following the operation. In one of these vision decreased from 20/200 to 7/200, and in the other it decreased from 20/50 to 20/50—. It remained unchanged in 5 cases and was improved in all the others. In 38 instances postoperative visual acuity of 20/50 or better was obtained.

Failures.—As in the study reported in the previous paper, the records of the cases in which the operation was unsuccessful (85) were analyzed, and an attempt was made to determine the cause of failure. In 12 of these cases no cause could be ascertained. The following unfavorable conditions were frequently encountered:

(*A*) Age: The relation between the age of the patient and the operative result is strikingly shown in table 2.

It is interesting to note that none of the 4 patients over 70 were benefited by operation.

(*B*) High Myopia: Among those patients with myopia of over 6 D. only 48 per cent were benefited by operation, while in the group with emmetropia or low hyperopia operation was successful in 72 per cent.

(*C*) Aphakia: Among the 13 patients with aphakia only 2 were benefited by operation. In each instance the detachment occurred after the removal of the lens. In 7 of these cases the operation was extraction of a hard cataract. Detachment followed discission for secondary cataract in 4 cases and came on in 1 case after needling for a congenital cataract and in 1 case after removal of a dislocated lens. Nine of the 11 patients in the group with aphakia in whom operation resulted in failure had separation of three fourths or more of the retina. In the 2 who were benefited approximately one half of the retina was involved.

TABLE 2.—*Relation Between Age and the Operative Result*

Age, Years	Cases Resulting in Cure, Percentage	Cases Resulting in Improvement, Percentage	Cases Resulting in Failure, Percentage
Up to 50	50.0	9.2	40.8
50 or over	28.6	6.3	65.1

(*D*) Hypotony: Operation helped only 11.7 per cent of those patients showing a preoperative intra-ocular tension of less than 10 mm. of mercury (Schiötz).

(*E*) Extensive Detachment: There was detachment of at least three fourths of the retina in 52.9 per cent of all the patients in whom operation resulted in failure.

(*F*) Multiple or Very Large Tears: There were multiple holes, large tears or extensive disinsertions in 38.8 per cent of the patients operated on unsuccessfully.

(*G*) Changes in the Choroid and Retina: As might be expected, degenerative changes in the choroid and retina were more frequently encountered among the cases in which operation resulted in failure.

Hemorrhage into the vitreous occurred postoperatively in 11 cases, and in 9 of these the retina remained detached. In 7 cases the retina was in position on the patient's discharge from the hospital but subsequently became redetached. The longest interval at which this occurred was two months. In this series 2 eyes came to enucleation. Severe

iridocyclitis necessitated the removal of one, while the other was lost as the result of endophthalmitis. In both instances one or more trephine openings were made in the sclera at the time of operation.

SUMMARY

In this series of 164 patients with retinal detachment 67 per cent were males and 33 per cent were females. The ages varied from 6 to 79 years.

A definite history of trauma was obtained in 17 per cent, while an additional 13 per cent gave an indefinite history of injury. A cure was effected in 62 per cent of the patients with a positive history of trauma.

Both eyes were found to be involved in 31 patients in the series.

Some degree of myopia was present in 53.8 per cent of the patients, 30.4 per cent of the group having myopia of at least 6 D.

Hypotony was found to be a grave prognostic sign.

In the patients having one half or less of the retina detached, improvement was obtained in 68.5 per cent, while in those with three fourths or more of the retina involved only 25 per cent were helped by operation.

The inferior portion of the retina was found to be the site of the detachment in 50 per cent of all the cases.

Those eyes in which no retinal holes were found responded to operation as well as those in which one or more retinal holes were located preoperatively. Widespread treatment of the affected area was used in all the cases.

In this group of 171 eyes the results of operation were: cure in 42.1 per cent, improvement in 8.2 per cent and failure in 49.7 per cent. Advanced age, high myopia, aphakia, hypotony, extensive detachment and multiple or large tears were frequently encountered among the cases in which operation resulted in failure.

INTRA-OCULAR NEUROFIBROMA

REPORT OF A CASE

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Neurofibroma within the eye has been so infrequently reported that every instance in which it occurs should be recorded. For this reason I wish to report the following case.

REPORT OF CASE

History.—B. D., a youth 21 years of age, was seen by Dr. J. H. Roth, of Kankakee, Ill., on Oct. 11, 1932. He gave a history of failing vision in the left eye, which had begun two years before, and of total loss of vision for the past year. Three weeks before examination the left eye became red and painful, and there was considerable tearing. The pain kept him awake at night. A general examination was not made.

Examination revealed the left eye deeply injected, the cornea hazy and the pupil widely and symmetrically dilated. There was no perception or projection of light. Vision of the right eye was 1.5—3. On October 20 the cornea was clearer, and it was thought a tumor could be seen within the eye on the temporal side. The tension was 59 mm. as measured by the Schiötz tonometer. The Wassermann test of the blood and urinalysis gave negative results. The following day, on examination in consultation with Dr. Harry Gradle of Chicago, a large tumor was visible on the temporal side of the fundus. Paracentesis of the anterior chamber to facilitate a better view of the fundus was advised. This procedure resulted in a hemorrhage into the anterior chamber, which cleared in two days, whereupon the tumor was again seen. The eye was enucleated on October 27.

Three and a half years later, attempts to find the patient for follow-up examinations were unsuccessful, as he had moved from the community.

Pathologic Examination.—The gross appearance of the bulb was normal. On transillumination a tumor was visible in the temporal quadrant.

After fixation the bulb was sectioned serially, each section being cut at 13 to 22 microns. Every tenth section was stained by Salzmann's hematoxylin-eosin technic, and other sections were stained with specific stains.

Microscopic Study.—The cornea was normal. Section 270 showed a subconjunctival paracentesis opening into which the angular line of the iris barely presented, the space remaining between the lips of the wounds and the conjunctiva being filled with a serofibrinous exudate similar to that filling the anterior chamber. This opening appeared to measure 0.75 mm. horizontally.

The peripheral fourth of the iris was adherent to the back of the cornea. There was a slight tendency for the iris pigment to curve onto the anterior surface of the iris at the pupillary border. The lens showed occasional small round droplets in the posterior cortex and was otherwise distorted, probably from fixing and sectioning.

The ciliary body was markedly thinned, and its processes were small and sparse.

The retina had been totally detached. The subretinal space was filled with a serum-like exudate and was intruded on by a tumor which had choroidal tissue reflected over it.

This tumor occupied a central position about 0.3 mm. temporal to the disk; it projected axially 10 mm. and was 14 mm. across in its broadest expanse as measured on the stained section. The base of the tumor adjoined the sclera, the outer layers of the tumor being more compact. It showed moderate vasculari-
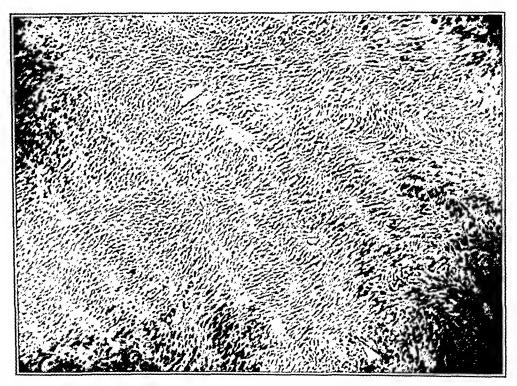
Fig. 1.—High power photomicrograph, showing palisading of cells, with cystic spaces and intercolumnar cells.

zation, the walls of the vessels consisting of a single layer of endothelium. These were not, however, true blood vessels, as no red blood cells were seen in most of them. The cells of the tumor were spindle shaped and were fairly regularly arranged, usually in columns or palisades, the long axis of the cells being perpendicular to the direction of the columns, many of which were thrown into irregular folds and whorls (fig. 1). The space between the columns contained an undifferentiated cytoplasm, no cellular membranes being distinguishable. Occasionally in these areas a spindle-shaped nucleus was seen, with its axis more or less parallel to the palisades. No mitotic figures were seen, and no melanotic cells were present except in the outermost layer adjoining the sclera or choroid, where the usual chromatophores were visible.

Serial section 180 showed, just outside the sclera, a cross-section of an artery, a large vein, a small vein and a nerve. The last was no longer characteristic

but was composed of a central area containing many nuclei, most of which were spindle shaped, having no regular arrangement and lacking any distinct separation from the surrounding tissue; these cells resembled the centrally lying spindle cells and faded out into a nearly normal appearing sheath of connective tissue

Fig. 2.—*A*, low power field, showing an artery and small vein and a cross-section of an abnormal ciliary nerve. *B*, high power view of the ciliary nerve shown in cross-section.

(fig. 2). Unfortunately, the sclera had been torn in the process of embedding and reembedding, because of air bubbles and sectioning, so that this group of struc-

tures could not be followed through the sclera. It is my opinion, however, that this is a part of intra-ocular formation which outside the eyeball had become plexiform in character. Other posterior ciliary nerves were seen in cross-section and longitudinally, but none showed this striking variation from the normal.

The optic nerve was normal; the retina had been torn from it during fixation.

A Perdrau stain of section 154 revealed a more or less dense net of argyrophilic fibers running mainly in a direction parallel to the palisades, where this formation was distinct, and elsewhere intermingling in a lacelike manner to form the supporting network. These fibers had processes extending like footplates to the walls of the vacuoles.

A diagnosis of intra-ocular perineurial fibroma, with secondary glaucoma and serous detachment of the retina, was made.

COMMENT

This neurofibroma, or perineurial fibroma, seems to have arisen from a ciliary nerve and showed changes similar to the plexiform neuroma, of which there are numerous reports. This single nonmalignant tumor of the nervous system has been named perineurial fibroblastoma because of its microscopic structure. However, the title which I have used is still the one in common usage. Penfield[1] defined a perineurial fibroblastoma as "an encapsulated tumor which arises from the sheaths of spinal nerve roots, cranial nerves, and peripheral nerves. It appears usually as a solitary tumor, much more often in a central location than a peripheral one. Tumors of the acoustic and spinal nerve roots far outnumber all others." His description of the microscopic details resembles closely that already given. He further stated that this tumor does not metastasize but may recur locally unless the segment of attached nerve is also removed. Its growth is usually slow. Sarcomatous degeneration has been reported but infrequently. Weil's[2] description is essentially the same as the foregoing one. He stated that formation of cysts is another feature of this tumor.

It may well be that the spaces described in my case are cystic changes rather than blood vessels. Indeed they were so interpreted for me by my colleague, Dr. Paul C. Bucy,[3] the neurologist. He stated:

This is a typical neurofibroma such as one sees on peripheral nerves or on the acoustic nerve, in spite of its unusual location. Sections impregnated by Perdrau's silver stain for reticulin are not entirely satisfactory but do reveal many fibers of reticulin among the spindle-shaped cells of the tumor.

The pathologic observations on a leukosarcoma of the iris by Zentmayer[4] were somewhat similar to those in the case reported here, as

1. Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 968.
2. Weil, A.: A Textbook of Neuropathology, Philadelphia, Lea & Febiger, 1933, p. 276.
3. Bucy, P. C.: Personal communication to the author.
4. Zentmayer, W.: Primary Sarcoma of the Iris, Removed by Iridectomy, Tr. Am. Ophth. Soc. 28:109, 1930.

the cells in his case were spindle shaped, arranged in bundles curling this way and that and had large blood vessels, the walls of which consisted only of endothelium. No mention was made of the presence of mitotic figures, and no silver stains were made. Roentgen therapy was utilized, and there was no recurrence of the tumor after three and a half years.

Two neurofibromas in one eye were described by Callender and Thigpen [5] in 1930. In their cases the tumors were "made up of spindle-shaped cells often arranged in whorls, occasionally having a palisade arrangement around a hyalin or fibrillar matrix. The ciliary tumor is somewhat more vascular and more cellular than the posterior one." My examination of a partial section of this eye (loaned by the Army Medical Museum) showed the tumor of the ciliary body to be somewhat similar to that in my case but with not nearly as characteristic an arrangement of the cells.

A flat neurofibroma of the choroid described by Freeman [6] in 1934 originated from the perivascular nerve plexus; it was not nodular in type, nor did its cells show palisading.

A case of melanosis of the uvea and melanoma of the iris, associated with generalized neurofibromatosis, has been described by Goldstein and Wexler.[7] This condition is an entirely separate one, more likely belonging in that large group of conditions known as von Recklinghausen's disease, whereas the tumor which I have described belongs with the solitary neurofibromas.

Papoleczy [8] described an intrabulbar and retrobulbar neurinoma arising from a posterior ciliary nerve, and Nitsch [9] reported two cases of neurofibroma of the eye. These reports were not available for this paper.

The discussion of the neurofibromas of the orbit has been purposely left out of this paper, but in a case of such a tumor, a recently reported one by Motto,[10] the structure was shown to be similar to that of the

5. Callender, G. R., and Thigpen, C. A.: Two Neurofibromas in One Eye, Am. J. Ophth. 13:121, 1930.

6. Freeman, D.: Neurofibroma of the Choroid, Arch. Ophth. 11:641 (April) 1934.

7. Goldstein, I., and Wexler, D.: Melanosis Uveae and Melanoma of Iris in Neurofibromatosis (Recklinghausen), Arch. Ophth. 3:288 (March) 1930.

8. Papoleczy, F.: Intrabulbar and Retrobulbar Neurinoma Arising from Posterior Ciliary Nerve, Arch. f. Ophth. 128:326, 1932.

9. Nitsch, M.: Neurofibromas of the Eye: Two Cases, Ztschr. f. Augenh. 69:117, 1929.

10. Motto, M. Paul: Neurofibroma of the Orbit, Arch. Ophth. 17:340 (Feb.) 1937.

tumor occurring within the eye in the case reported here. The tumor in Motto's case may also have originated from a ciliary nerve, but it was so far back that it did not grow within the bulb.

It is my belief that a number of the leukosarcomas which have been described in the past belong in the group of neurofibromas. As an

Fig. 3.—Slender spindle-shaped tumor cells, which are arranged in parallel rows. All the spindles are free from pigment; in this illustration they are shown dark because the author (Schieck) was interested less in the cells as such than in their arrangement in rows (from Schieck,[11] fig. 18).

v. Graefe's Archiv. Bd. LXXVII.

Fig. 2.

Fig. 1.
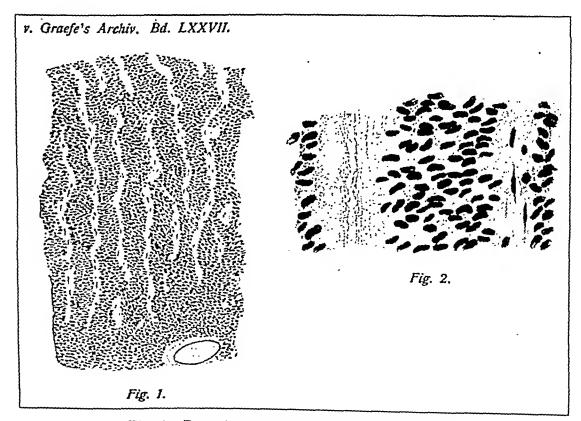
Fig. 4.—Reproduction of figures 1 and 2 of Fuchs.[12]

example, in his book on melanosarcoma of the uveal tract F. Schieck [11] described a tumor (illustrated by his figure 18) similar to this one

11. Schieck, F.: Melanosarcoma of the Uveal Tract, Wiesbaden, J. F. Berg-mann, 1906.

except that there were chromatophores and pigmented spindle cells in the outer layers. However, these pigment-bearing cells could well have been normal choroidal pigment cells caught up in the tumor.

E. Fuchs [12] reported a similar case (fig. 4). His description, in part, is as follows:

> The nuclei are in long bands lying parallel to one another, with their long axis vertical to the axis of the bands. The nuclei are imbedded in a homogeneous interstitial substance in which no cell borders are distinguishable so that perhaps there is a kind of syncytium. The nuclei are not lying bare to one another but in irregular rows behind one another; on the number of these rows, varying from 2 to 10, depends the breadth of the nuclear bands. The interstitial space between two bands is filled with a homogeneous substance which is usually vacuolated (frequently such is found between the nuclei in the bands and so appear to be an artefact developing through hardening). A clear framework between the bands is not seen. In one case in the interstitial substance between the nuclear bands are single round nuclei; in another case single long nuclei the axis of which is vertical to those of the bands and are perhaps connective tissue. Occasionally between the bands are single pigment granules, though usually the tumor is not pigmented. The sparse blood vessels are not directed towards the nuclear bands; they cut crosswise to them very frequently. I have observed this structure in three cases, in one case in the entire tumor, in two others in single sections.

·I believe that with the special stains of today these tumors would be found to have originated from nervous tissue.

SUMMARY

A case of intra-ocular neurofibroma or perineurial fibroblastoma has been described. The outstanding characteristics were the regular' arrangement or palisading of the cells, the presence of reticulin fibers, cystic changes and the absence of pigment and of mitotic figures.

A review of other intra-ocular neurofibromas as compared to this one is given, showing that some tumors described in the past under the title of melanosarcoma and leukosarcoma may well belong in this group.

A discussion of the neurofibromas of the orbit has purposely been excluded in this presentation, though it is readily seen that such a tumor as I have described arising from a ciliary nerve may be the intra-ocular expression of changes which could also affect only the orbital portion.

12. Fuchs, E.: Sarcoma of the Choroid, with Remarks Concerning Necrosis of the Uvea, Arch. f. Ophth. **77**:304, 1910.

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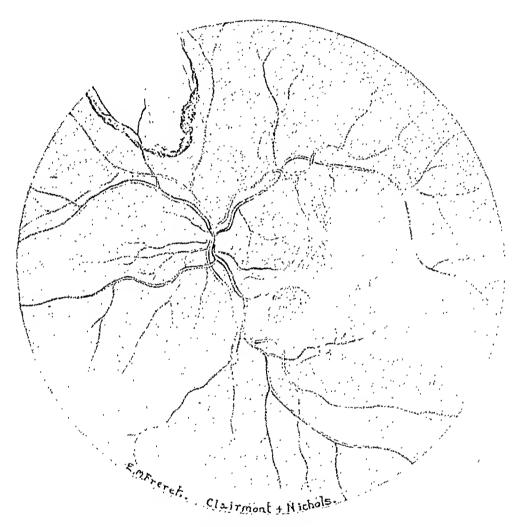
CASE 2.—J. F., a man aged 39 years, was seen on Feb. 1, 1937. He stated that his right eye had become affected eight years before. He received no treatment and could give little information about his case. Vision with a glass was 20/200. The vitreous was clear. There was a preretinal organized opacity in the vitreous in the macular region, which was not connected with the large atrophic choroidal scar, extending downward and outward. The opacity in the vitreous was fixed, anterior to the retinal vessels and had the same irregular, branching structure noted in the other cases. (This condition is illustrated in the figure.)

Preretinal organized vitreous opacity.

CASE 3.—C. S., a youth aged 17, was seen on Oct. 15, 1935. He said that the sight in the right eye became affected in June. On examination vision in the right eye was 8/200. There were many membranous opacities of the vitreous; a stationary opacity was situated in front of the macular region, and another partly covered the disk and extended down in front of the inferior retinal vessels. A large active choroidal lesion was present in the lower and outer periphery. As the patient reacted to tuberculin, he was put on a regimen of treatment with tuberculin, sunlight baths, rest and tonics. On Jan. 7, 1936, vision had improved to 20/50 —. The vitreous changes had diminished and now consisted of a branching opacity extending across the optic disk and adherent to the retina in places. The choroidal focus was healing. After spending the rest of the winter

on a ranch in Arizona, with continuation of treatment with tuberculin, he had vision of 20/20 — on April 6. The opacity of the vitreous had largely cleared, and the choroidal area had healed. A curvilinear opacity was noted in the vitreous, with broad, branching extensions adherent to the retina. There was a connection with the optic disk and some of the vessels just below, though no vessels could be seen entering the opacity, and in no place was there a suggestion of a detachment of, or even traction on, the retina.

COMMENT AND SUMMARY

This clinical picture consists of a large choroidal patch with the corneal deposits and vitreous opacities usually present in cases of acute choroiditis. As the process heals the vitreous clears except for a curious fixed opacity which is adherent to the retina, particularly in the macular region, and is not connected with the area of choroidal atrophy. The opacity becomes very faint in places, and surprisingly good vision is sometimes obtained. No new-formed vessels pass into the opacity, and there is no suggestion of detachment of or traction on the retina.

PATHOGENESIS OF DISCIFORM DEGENERATION OF THE MACULA

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In 1875 Pagenstecher gave a description of the macroscopic and microscopic appearance of a macular lesion observed in an eye after its removal for adherent leukoma and secondary glaucoma. His descriptions and illustrations of this lesion leave little doubt that the eye was affected with what is now known as disciform degeneration of the macula. His is the first case of this disease that we were able to find recorded in the literature. He designated the condition chorioidio-retinitis in regione maculae luteae. The first employment of the term degeneratio maculae luteae disciformis was by Oeller in 1905. In 1926 Junius and Kuhnt adopted this term, substituting, however, *Netzhaut-mitte* for macula lutea, and definitely established the condition as a disease entity. Other terms to designate the disease, employed prior to the publication of their monograph, are indicated by the titles in the appended bibliography. The term favored by Junius and Kuhnt, although in several respects misleading, has now been generally accepted. Following is an analysis of all the cases of senile disciform degeneration at the macula reported in the available literature. There were eighty-four such cases, one hundred and twenty-nine eyes being affected. Thirteen of these eyes were examined microscopically. The three cases to be described in this communication are not included in the analysis.

ANALYSIS OF RECORDED CASES OF SENILE DISCIFORM DEGENERATION OF THE MACULA

Race.—No patient was stated to be other than white, but only a few reports specified the race.

Frequency.—There is no doubt that the cases described in the literature represent only a fraction of the total number of cases recognized as instances of disciform degeneration at the macula. Kahler and

O'Brien stated that twelve cases were observed in the ophthalmologic clinic of the State University of Iowa in one year.

Age.—In the majority of cases the condition occurred in the fifth and sixth decades of life. The youngest patient was aged 39 (Neame); the oldest was 83 (Kahler and O'Brien). The average age for the entire group was 68. Age, therefore, was a definite factor.

Sex.—Of the eighty-four patients, forty-seven were male and thirty-four female; the sex of three was not stated.

Trauma and Inflammation.—There was no evidence that these were factors in the disease.

General Condition.—Many of the reports contained little information other than a description of the ocular lesion. Nineteen of the patients were reported to have general hypertension. Fifteen had general arteriosclerosis. Eight had a cardiac condition, and four were nephritic. Four were diabetic. Only three were syphilitic. One patient was known to have lived eleven years after the ocular condition was first observed. Eight patients died of coronary disease.

Eye Affected.—In thirty-nine cases the condition was unilateral, the right eye being affected in eighteen. Among twenty-one of the cases in which the condition was bilateral it was determined that the right eye was the first affected in twelve.

Symptoms and Mode of Onset.—Central vision in the affected eye was in all cases greatly impaired or completely abolished when the patient first came under observation. Some patients noted a positive central scotoma. No important improvement in vision occurred in the progress of the disease in any case. As to whether the disease had the same mode of onset in all cases, and, if so, whether this was gradual or sudden, it was impossible to determine. The fact that only gradual loss of vision was noted by most patients may have been due to preceding development of senile macular changes which prevented any subjective recognition of the onset of disciform degeneration, even if this onset was actually abrupt. In a few cases the patient noted disturbances in vision such as loss of visual acuity, metamorphopsia and photopsia before any definite lesion could be seen with the ophthalmoscope, but here again it is not clear that these were prodromal symptoms of disciform degeneration. On the other hand, when a patient stated that his loss of vision was sudden, it may actually have been gradual and only suddenly discovered. The only positive evidence regarding the mode of onset was that furnished by twelve eyes. In each of these the onset was undoubtedly sudden, since a subretinal hemorrhage was observed at the first ophthalmoscopic examination.

Aside from Pagenstecher's case, in which there was glaucoma secondary to an adherent leukoma, glaucoma was not known to have developed in any case.

Ophthalmoscopic Signs.—In twelve eyes there was first observed a subretinal hemorrhage in the macular region, which elevated the retina in the form of a mound. Adjacent to this were other hemorrhages. These, which were usually described also as subretinal, often outlined the ultimate mass. More remote hemorrhages were apt to be described as occurring within the retina. These were observed to disappear, and new ones were seen to occur in other places. In cases, comprising the majority, in which the lesions were first observed at a later stage, hemorrhages were less frequently noted. However, in seventy-five of the one hundred and twenty-nine eyes hemorrhages were noted, and in thirty-six eyes hemorrhages adjacent to the mound were still in evidence.

The mound as seen early was described as grayish, greenish or almost black. Later it became white except for more or less deposition of pigment. The retinal vessels always were visible in front of the mass. In several cases arteriovenous communications were noted. The size of the mound varied from one-half to several times the size of the optic disk. In some cases its height was considerable, in one case reaching 6 D. In some cases it was dimpled over the hypothetic macula. It was usually more elevated early in the progress of the disease and often appeared to "settle" as the disease progressed.

The margins of the mound were usually distinct. Almost always at least a portion of the edge was sharply etched against the adjacent retina. The shape was often disciform but sometimes assumed other contours. The location of the mass was the macular region between the superior and the inferior temporal vessels. Rarely did the lesion exceed the limits of these vessels.

Associated Fundic Changes.—Sclerosis of the retinal vessels was recorded as existing in only twenty-five out of the one hundred and twenty-nine affected eyes. In each of the five cases in which the condition later became bilateral the second eye showed senile macular changes before the development of the disciform lesion. These senile changes included drusen, pigmentary changes, and white spots in the papillomacular region and along the macular vessels. Among thirty-seven cases in which the condition was unilateral the fellow eye showed senile macular changes in fifteen and was reported normal in only sixteen (four eyes had senile cataract, which prevented examination of the fundi, and two had been enucleated for other causes).

In sixty-one eyes some type of white or yellowish white spots of degenerative or exudative character were found near the lesion at some stage of its progress. These ranged from small spots the size of a pinpoint deep in the retina to frank circinate retinitis (seen in twenty-two eyes). In two of the cases of the latter condition, circinate retinitis was observed first; in one of these two, disciform degeneration was found in addition fourteen months later, and in the other, a few months

later. In the other cases circinate and disciform lesions were seen simultaneously, and it was impossible to determine which condition preceded the other. In a case (reported by Spicer in his discussion of Lawford's paper) not included in this series, circinate retinitis existed in the fellow eye, and we have recently seen another such case. In the description of one case, which was briefly reported by Neame, it was stated that angioid streaks were present in the other eye. In a case in which the condition was unilateral, reported by Davis and Sheppard, which will be referred to later, angioid streaks were present in each eye. Feingold stated that "similar changes in the macula were observed in a patient showing all characteristic fundus changes of angioid streaks," but gave no further details regarding the eye.

Microscopic Observations.—Thirteen eyes were examined microscopically. One eye was removed for glaucoma that had occurred secondary to an adherent leukoma; four were removed for suspected tumor, and eight were removed post mortem. The observations can be briefly summarized as follows:

1. The retina over the mass was observed to show degenerative and atrophic changes in all but two cases. These were believed by most observers to be secondary changes.

2. The pigment epithelium in all the cases was seen to be markedly proliferated and was a prominent component of the mound.

3. In six of the eyes examined microscopically the lamina vitrea was observed to show pathologic changes. Michel, Verhoeff and Behr noted colloid excrescences on it, as well as variability in its staining properties and thickness. Six eyes were seen to show breaks in Bruch's membrane through which blood vessels, red blood cells or lymphocytes passed. In four instances this membrane was reported to be intact and normal.

Other than showing signs of compression, the choroid of eight eyes was observed to be completely normal. Elschnig noted atheromatous choroidal changes. Verhoeff reported the choroidal vessels to be slightly sclerotic. Hanssen noted small collections of lymphocytes in the choriocapillaris in the region of the lesion, and Behr observed proliferation of the intima of the middle-sized arteries.

The mound was uniformly reported to consist of proliferated pigment cells and chiefly of fibrous tissue. Behr reported strands of elastic tissue, which he believed lifted off from the outer fourth of the lamina vitrea. Small amounts of blood and albuminous exudates in various stages of organization were occasionally observed. Vessels presumably chiefly from the choroid were observed within the mound in all cases, and occasionally a capillary was observed to enter it from the retina. Choroidal vessels were seen by several observers to penetrate the lamina

vitrea. Michel noted cartilage within the pseudotumor, and Axenfeld reported an area of ossification within the mass.

Pathogenesis.—Elschnig placed the origin of this disturbance in the retina and its vessels, and Junius and Kuhnt, in the inner layers of the retina; Ormond, Duynstee and others believed that the disease originated from the capillaries of the outer retinal layers.

Michel contended that the origin of the growth was in the lamina vitrea. Behr evolved a theory of vital functions which he attributed to the lamina vitrea and contended that a pathologic disturbance of this membrane interferes with these functions and stimulates hyperplasia.

Hanssen, who noted lymphocytic infiltration of the choriocapillaris, concluded that the choroid was the chief offender.

Pagenstecher, Axenfeld, Hegner, Neame, and Coppez and Danis subscribed to an inflammatory cause.

Oeller, Batten, Beatson and Knapp believed that the mass originated from a subretinal exudate or hemorrhage or both.

Possek thought that the exudate was caused by arteriosclerotic changes in the tissues of the eye. Sclerotic changes in the choroidal vessels particularly were observed and emphasized as a possible cause of nutritional disturbance with resultant hyperplasia by Cords, Pallarés, Verhoeff and Paul.

JUVENILE DISCIFORM DEGENERATION OF THE MACULA

In the literature there have been described cases in which a macular condition resembling senile disciform degeneration has occurred in persons below the age of 40 years. For convenience we shall refer to it as juvenile disciform degeneration of the macula. It differs from the senile type also in the fact that the lesion tends to heal, leaving few, if any, changes and slight or no impairment of vision. Just how frequently it occurs is problematic, because it may often have been mistaken for some other condition. Special attention was called to this condition by Junius in 1929. He reported four cases under the classification juvenile retinitis exsudativa macularis, three of which seem to us to belong to the type under consideration. The ages of the patients were 34, 32 and 27 years, respectively, and only one eye was affected in each case. In the first case the condition reached its height in ten weeks, and vision was then reduced to counting of fingers at 2 meters. In six months the lesion had entirely disappeared and vision was normal. The patient was seen again twenty-four years later, at which time the fundus and vision were still normal. In the second case there were two small hemorrhages at the margin of the lesion. Vision was reduced to perception of movements of the hand. At the end of two months the lesion had largely disappeared, leaving a pale oval area partially demarcated by a fine pigmented line, and the vision was 0.3. In the other case, the

onset was sudden, and vision was reduced to the counting of fingers at 0.75 meter. Whether vision later improved was not determined, because the patient was lost sight of. The patient was subject to ophthalmic migraine. The remaining case reported by Junius (his case 3) seems to us to belong in an entirely different category. In this instance there were within the retina of each eye extensive white exudate around the optic disk and a massive white star figure around the fovea. The patient was aged 14 years. The only important similarity that the condition in this case had with that in the others lay in the fact that the exudate disappeared and there was little residual impairment of vision.

In 1935 Davis and Sheppard reported two cases as belonging to the type described by Junius. In one case the age of the patient was 23 years. "In each macular region was found an oval golden yellow area over which the retinal vessels passed." Visual acuity was 20/20—2 in each eye. Whether the lesion was elevated was not stated. Two years later, changes could still be seen in each macula, and vision of the right eye was 20/100 and that of the left 20/30. In their second case an elevated disciform lesion was observed in the right eye of a patient aged 41 years, who had angioid streaks in each eye. The observation was made about two years after central vision was said to have been lost. No improvement in vision was noted. It seems to us that the condition in this case belongs to the senile type of disciform degeneration, and we have therefore included it in the list of cases just analyzed. The assumption of Davis and Sheppard that the loss of central vision coincided with the onset of the disciform lesion is not necessarily correct, since sudden loss of central vision is a typical occurrence in cases of angioid streaks.

Junius regarded the lesions in his cases as retinal in origin and due to some vascular disturbance, but concluded that their etiology was still in the dark.

From the foregoing review of the literature it is obvious that the pathogenesis of senile disciform degeneration of the macula has not hitherto been determined. The literature relating to the juvenile type we have not yet fully examined, but thus far we have been unable to find in it any suggestive evidence as to the pathogenesis of the ocular lesions. The following cases studied by us seem definitely to reveal the pathogenesis of the senile, and strongly to indicate that of the juvenile, type of the disorder.

REPORT OF ADDITIONAL CASES

CASE 1.—*History.*—Miss G. L., aged 49, a white woman, first consulted Dr. Verhoeff on April 6, 1931, complaining that four days previously she discovered that the sight of the right eye was blurred.

Ocular Examination.—Vision of the right eye with a $+2.50$ D. sph. was 5/200, and that of the left eye with a $+3.75$ D. sph. $\subset$ -1.00 D. cyl., ax. 125 was 20/15. On ophthalmoscopic examination of the right eye there was found occupying the macular region and extending farther below than above an almost black moundlike elevation with a smooth surface. Extending beneath the retina from the margin of the mound were several extravasates of blood. The fundus otherwise appeared normal—there was no sclerosis of the retinal vessels. The fundus of the left eye was normal.

General Condition.—The patient stated that her general health was excellent. Her urine was free from sugar and showed only an insignificant trace of albumin.

Course and Outcome.—A diagnosis of sarcoma of the choroid was made, and on April 9 the right eye was removed with the area under local anesthesia and a glass ball implanted.

On May 8, 1936, vision of the left eye with correction was 20/15, and the fundus was still normal. The general health of the patient remained excellent.

Pathologic Examination.—The eye was fixed in a 4 per cent solution of formaldehyde for forty-eight hours, and this was followed by immersion in acid alcohol for twenty-four hours and embedding in pyroxylin (celloidin). Serial sections showing the lesion in the fundus were made and stained with hematoxylin and eosin and with Verhoeff's stain for elastic tissue.

Microscopic Observations: The eye was normal except for the lesion in the macula and the presence of an unusual number of eosinophilic cells in the stroma of the iris. These cells (apparently eosinophilic myelocytes) were large, had small, round, solidly staining nuclei and were packed with fine eosinophilic granules. As many as five of these cells were sometimes observed in the high power field. The iris contained no plasma cells or other inflammatory cells aside from the eosinophils. The vessels of the retina and choroid and the posterior ciliary vessels were notably free from sclerosis. Peripheral cystoid degeneration of the retina was very slight. The supposed tumor of the choroid was seen to be a large mass of fresh blood beneath the retina in the macular region (fig. 1). This raised up the retina in the form of a mound measuring 1.5 mm. in height and 6 mm. in diameter. The mass was somewhat eccentric with reference to the fovea. It began 2 mm. to the temporal side of the fovea and extended to the margin of the disk. It reached its greatest height at a distance of 4 mm. from the disk, somewhat above the fovea. At least two-thirds the total mass of blood was beneath the pigment epithelium, which it had separated from Bruch's membrane in the form of a vesicle. The vesicle was solidly filled with blood and was 4 mm. in diameter. Its inner margin was 2 mm. from the margin of the optic disk, and its center was somewhat above the fovea. The vesicle was everywhere intact, except on the nasal side, where in a few sections it showed a rupture about 0.25 mm. wide through which blood had extended outside the vesicle between the retina and pigment epithelium. In the region of the fovea the wall of the vesicle was still in contact with the neuroepithelium of the retina in an area about 2 mm. in diameter, except immediately behind the fovea, where there was a slight amount of serum. In contrast to the blood within the vesicle, the blood outside of it contained lakes of serum free from blood cells, indicating that it was under less pressure than was originally the blood within the vesicle. The choroid, including the membrane of Bruch, was normal and intact except in a small area at, and just outside, the lower outer margin of the vesicle. Here the source of the hemorrhage was apparent (fig. 2). In an area 0.6 mm. wide the choriocapillaris with the membrane of Bruch was

EXPLANATION OF FIGURES 1, 2, 3 AND 4

Figure 1 (case 1) is the photomicrograph of a section showing hemorrhagic extravasate in the macular region beneath the pigment epithelium. The latter has been outlined with ink to show its position more clearly. In this section it was intact except for obviously artificial breaks. The blood outside of the vesicle extending to the margin of the disk left the vesicle through a small break in its wall seen in other sections. At the summit of the mound is a small amount of serum between the pigment epithelium and the retina proper. Magnified × 9. Figure 2 (case 1) is the photomicrograph of a section showing rupture of the choriocapillaris at the periphery of the mound, and the pigment epithelium elevated by hemorrhagic extravasate. Other sections showed this extravasate continuous with that forming the main mass on the left. Magnified × 48. Figure 3 (case 2) is the photomicrograph of a section showing the mound in the region of the fovea. The pigment epithelium in the posterior part of the mound is not the original pigment layer but has been newly formed. It is widely separated from Bruch's membrane by connective tissue. Magnified × 13. Figure 4 (case 2) is the photomicrograph of a section showing the nasal two thirds of the mound and large masses of old blood undergoing organization. The strands of pigment epithelium are taking part in the organization of the mass. Bruch's membrane is intact. Magnified × 44.

Figure 1

Figure 2

Figure 3

Figure 4
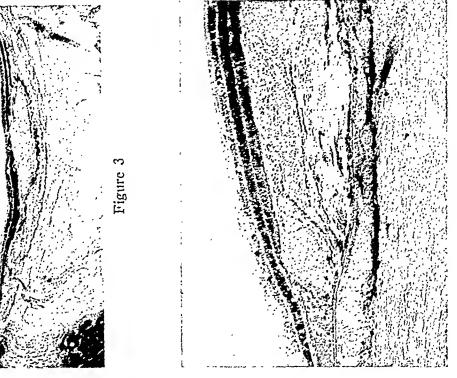
torn up in a ragged manner by an extravasation of blood which raised up the pigment epithelium in the form of small vesicles and then extended into the large vesicle described. From this point for a short distance beneath the large vesicle the choroid showed slight infiltration with lymphocytes and slight exudation of the latter cells into the mass of blood. The mass of blood here contained a slight amount of fibrin. From the margin of the vesicle outward the extravasate extended within the choroid just beneath the choriocapillaris for a distance of about 3 mm. Elsewhere the choroid was free from infiltration and hemorrhage. The retina proper was wrinkled in and around the fovea, probably as a result of the embedding process but otherwise appeared perfectly normal. It showed no exudation, hemorrhages or edema.

CASE 2.—*History.*—Daniel M., aged 77, a married white man, was admitted to the clinic of the Massachusetts Eye and Ear Infirmary on Jan. 15, 1936, complaining of blurred vision of his left eye.

He recalled a slight decrease of visual acuity of his left eye in the summer of 1934. He was certain that in 1935 vision of the same eye was much less clear than that of the right eye. In November 1935 he was told by an optometrist that his left eye was "stone blind."

Ocular Examination.—Vision of the right eye with a + 0.75 D. sph. was 20/30. Vision of the left eye was less than 20/200. Each eye was white and quiet. Arcus senilis was present. The corneas and other media were clear. The pupils reacted normally to light and in accommodation.
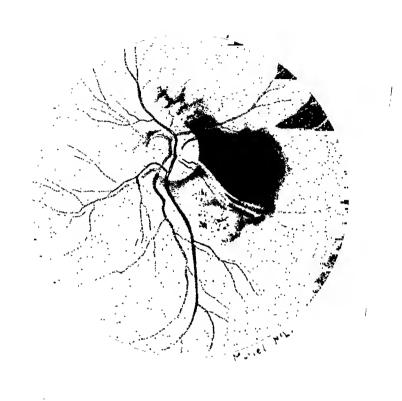
Examination of the Fundus.—The optic disk of the right eye was normal. In the macular region of this eye there were many colloid excrescences (Tay's choroiditis). The fovea was sharply outlined. The retinal vessels showed no definite sclerosis. The fundus was otherwise normal. The optic disk of the left eye was normal in color. The retinal vessels of this eye showed slight, if any, sclerosis. Adjacent to the temporal margin of the disk there was a large elevated mound, almost black, about 2 to 3 disk diameters in size, clearly demarcated and surrounded by a band of deep hemorrhage (color plate 1). The retinal vessels coursed in front of the mass and, with the exception of one vessel and its two branches, appeared normal. This vessel, which extended from the edge of the optic disk, became white as it crossed the mound, as did also its two branches. There were two diffuse whitish areas adjacent to the mass, one at its lower margin and the other at the upper border of the disk, each containing hemorrhagic spots.

At this time a definite diagnosis of hemorrhage behind the pigment epithelium was made.
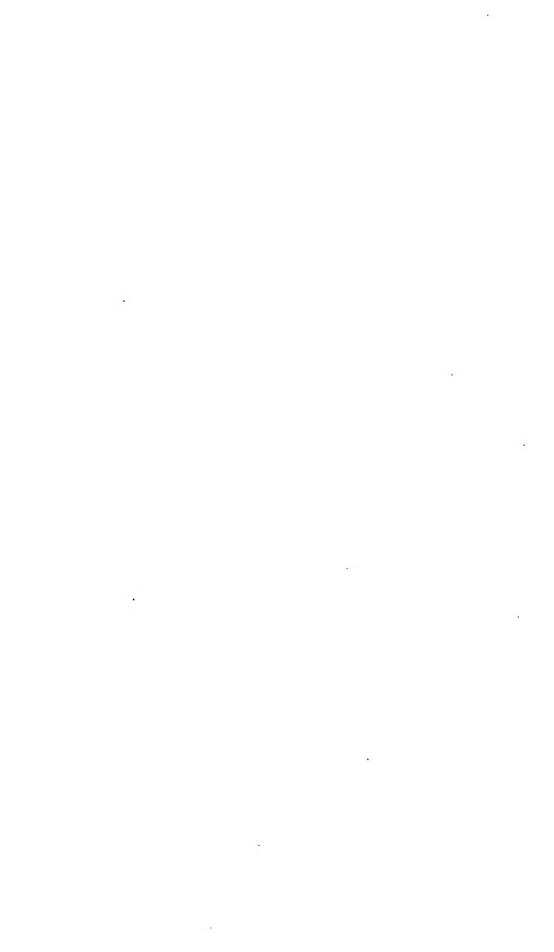
Physical Examination.—The blood pressure was 140 systolic and 80 diastolic. The heart was normal.. The radial and dorsalis pedis arteries were not palpable. The urine contained no albumin, sugar or casts and rare red blood cells and white cells. The Wassermann and Hinton tests were negative. The blood count showed 75 per cent hemoglobin, 4,640,000 red cells and 8,000 white cells, with 63 per cent polymorphonuclears, 30 per cent lymphocytes, 1 per cent eosinophils and 6 per cent monocytes. The platelets were normal. Slight achromia and variation in size of the red cells were present.

Course and Outcome.—The patient was observed every two weeks during the next three months.

An area immediately adjacent to the temporal edge of the mound early became more shallow than the rest of the lesion and ultimately flattened out to the retinal level. The reflex from this portion became red. At the end of six weeks this

Color plate 1 (case 2).—Ophthalmoscopic appearance of the macular lesion when first seen. The picture is practically identical with that seen in case 1, except that in the latter case the retinal opacity below the mound and that above the optic disk were not present.

Color plate 2 (case 2).—Ophthalmoscopic appearance of the lesion three months later. The mound has become almost completely white. The hemorrhagic extravasates around it still persist, as do also the two retinal opacities. The latter were observed to be due to serum beneath the retina. The apparent minute retinal hemorrhages here were seen to be collections of red corpuscles in this serum.

portion of the mound seemed to disappear, leaving normal retina in its place except for a few deep hemorrhages.

Within four weeks the lesion became white in its temporal portion, gradually shading into gray and dark gray toward its nasal edge. The temporal edge was always sharply raised. At ten weeks the lesion was entirely white except for a small gray edge at its upper border. There were a few heavy granules of black pigment near its outer margin.

The lesion throughout its course remained completely surrounded by a band of deep subretinal hemorrhage. The white areas at the upper border of the disk and the lower border of the lesion persisted. They were raised at first, but finally became flatter, more pink and less distinct. In the vessels that originally appeared white as they crossed the mound, blood became visible in six weeks, and they then assumed the same normal appearance as the other retinal vessels. The final appearance of the lesion at the end of three months is depicted in color plate 2.

Three months after the patient was first observed, of his own volition he consulted two other ophthalmologists. Both these advised removal of the eye because it was practically blind and because of the possibility of a malignant growth. The patient was given our opinion that the condition was not malignant, but did not receive the positive assurance that he desired. He requested removal of the eye, and it was therefore removed, on April 15, 1936.

Pathologic Examination.—Preparations were made as in case 1.
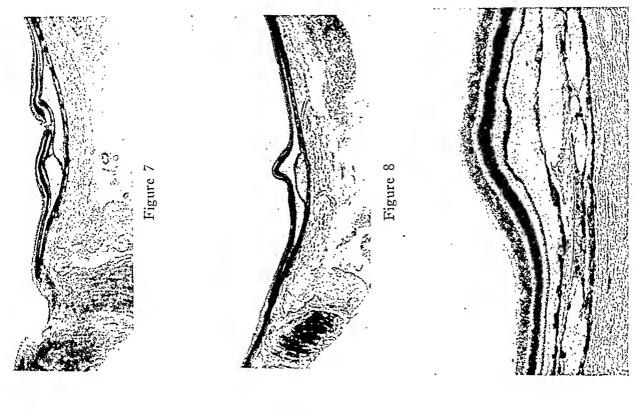
Macroscopic Observations: The vitreous was clear and was not separated from the fundus or the lens. There were slight senile pigmentary changes at the ora serrata. In the macula there was an area 4.5 mm. in its greatest diameter, elevated about 1.25 mm. It was roughly circular but showed some indentations of the margin. Its surface showed slight grayish mottling and, in the center, the normal yellowish color of the macula, but otherwise appeared almost white. Around the area was a dark border from 0.25 to 0.75 mm. wide. Radiating from near the center were many fine white lines (artificial wrinkles?) which extended into the dark border. The retina otherwise was in situ everywhere.

Microscopic Observations: Aside from the lesion in the macula, the eye showed no pathologic changes and unusually slight senile changes. The ciliary processes showed only slight hyaline change. The two layers of epithelium of the pars plana showed fairly marked proliferative changes in some sections. There was almost no peripheral cystoid degeneration of the retina, and colloid excrescences were not seen anywhere. An unusual observation was that the myelinization of the fibers of the optic nerve ended about 1 mm. behind the lamina cribrosa. The optic disk was normal. The vessels of the iris, ciliary body and retina and the posterior ciliary vessels were normal. The vessels of the choroid were also notably free from endovasculitis, although an occasional artery showed slight intimal proliferation—less than would be expected, considering the age of the patient. The lesion in the macula (fig. 3) was seen to consist of a mound of finely fibrillated connective tissue permeated with blood and containing large masses of blood in the process of organization. It was situated between the retina proper and Bruch's membrane, was thickest behind the fovea and gradually became thinner towards the periphery all around. It reached to about 0.5 mm. from the margin of the optic disk. The new tissue was everywhere closely applied to Bruch's membrane, which, except for a few small defects to be described, was intact beneath it. The tissue in places was stained lightly by eosin but more often was stained slightly bluish by hematoxylin, especially near the choroid. The largest masses of blood were situated in the upper part of the mound and were nearer the retina than the choroid. In sections they comprised about half the

EXPLANATION OF FIGURES 5, 6, 7, 8 AND 9

Figure 5 (case 2) is the photomicrograph of a section showing a vein leaving the mound near its periphery and joining a large choroidal vein through a break in Bruch's membrane. Magnified ✕ 54. Figure 6 (case 2) is the photomicrograph of a section showing an artery entering the mound from the choroid through a break in Bruch's membrane. Magnified ✕ 87.

Figure 7 (case 3) is the photograph of a section showing the pigment epithelium elevated in the form of a vesicle by serous exudate in the foveal region. Serum has exuded from the vesicle and collected beneath the retina proper. Magnified ✕ 10. Figure 8 (case 3) is the photomicrograph of a section showing the vesicle near its periphery. It contains two collections of red blood corpuscles. Here there is beneath the retina a considerable amount of serum which has been exuded through the epithelial walls of the vesicle. Magnified ✕ 9. Figure 9 (case 3) is the photomicrograph of a section showing formation of connective tissue between the pigment epithelium and Bruch's membrane at the periphery of the vesicle. This tissue is entirely mesoblastic in origin and derived from fibroblasts that have penetrated Bruch's membrane. Magnified ✕ 45.

Figure 7

Figure 8

Figure 9

Figure 5
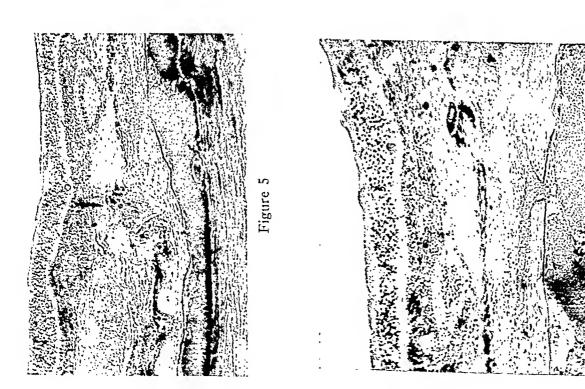
Figure 6

thickness of the mound (fig. 4). The tissue was fairly rich in fixed cells but was free from lymphocytes, plasma cells, pus cells and macrophages. It contained no hematogenous pigment, fatty acid crystals or cholesterol. The original pigment epithelium could be identified only at the periphery of the mound. In the central portion of the latter no pigment epithelium, new or old, remained adherent to Bruch's membrane or to the outer surface of the retina. At the periphery all around, connective tissue continued from the mound as a thin layer between the original pigment epithelium and Bruch's membrane for a distance of about 0.5 mm. As the single layer of pigment epithelium extended toward the center of the mound it was seen to have proliferated into two main layers which had diverged from each other and then further proliferated in an irregular fashion. Some sections showed a large central area of the mound entirely surrounded by a single layer of pigment epithelium. Posteriorly the latter had here undergone complete necrosis and appeared as an eosinophilic granular membrane free from nuclei but still containing pigment. In places, especially at the periphery, but also elsewhere, the pigment epithelium had proliferated in the form of irregular strands, some of which suggested acini. In the vicinity of these strands the connective tissue tended to assume a more hyaline character than elsewhere. Here there were also spindle cells, some of which contained more or less pigment, others being free from pigment. The epithelial origin of many of the latter was evidenced by their continuity or contiguity with the pigment cells and their close resemblance to the latter. From this evidence it was clear that a large proportion of the cells in the mound were derived from pigment epithelium. Many such cells were seen within the masses of blood and apparently were playing a large part in the process of organization. The mound contained a few thin-walled blood vessels, most of them slightly larger than capillaries, a few about three times this size. These were seen chiefly near the choroid and at the periphery. No vessels were observed entering the mound from the retina, but after careful search two fairly large vessels were noted extending into it from the choroid through sharp breaks in Bruch's membrane (figs. 5 and 6). One of these was directly connected with a large choroidal vein. In addition, a few microscopic breaks were noted in the membrane, through which fibroblasts extended into the mound from minute collections of newly formed fibroblasts in the choroid. In places Bruch's membrane was slightly wrinkled, evidently owing to contraction of the tissue forming the mound. Aside from these changes and slight compression, the choroid, including the choriocapillaris, appeared perfectly normal behind the mound, as it was elsewhere. The source of the hemorrhage was no longer recognizable.

The retina over the mound was relatively little altered. The nuclear layers, the ganglion cells and the layer of nerve fibers appeared normal. There were no hemorrhages, edema or exudation, and the vessels were normal. The outer surface was distorted and occasionally folded in the form of pseudorosettes. The rods and cones had been largely destroyed but were often still recognizable. The external limiting membrane was almost completely intact. As has already been stated, the pigment epithelium had completely disappeared from beneath the retina over the central portion of the mound, and the new connective tissue was directly applied to the neuroepithelium. In the fovea was a minute break into which the connective tissue extended.

Almost everywhere around the mound was a layer of blood extending between the retina and the pigment epithelium for about 1 mm. and on the nasal side reaching the margin of the optic disk. Into this blood many pigment cells had migrated. Unlike the blood within the mound, it was not undergoing organization.

Sections passing through the retinal opacity below the mound and others passing through that above the disk showed serum instead of blood between the retina and the pigment epithelium.

CASE 3.—*History.*—David W., aged 63, a married white man, was admitted to the clinic of the Massachusetts Eye and Ear Infirmary on Nov. 19, 1935, complaining of blurred vision in the left eye. He had noticed a black spot (probably of no significance) before the left eye for about five months and double vision with the eye for about two weeks. He had always been well except for measles in childhood and influenza in 1915. The family history was unimportant.

Ocular Examination.—The right eye was normal and had vision of 20/15 with a + 2.00 D. sph. ⊃ + 0.75 D. cyl., ax. 90. The optic disk showed an unusually deep and wide physiologic cup. The vision of the left eye with the glass the patient was wearing was 20/100, but with a + 1.00 D. sph. added it was 20/30. The retina showed in the macula a grayish elevation about the size of the disk. The retinal vessels were normal. The disk was cupped as in the right eye. There were no retinal hemorrhages. The vitreous was clear. On November 22 vision of the left eye with a + 1.50 D. sph. added to the glass was 20/30. On December 13 vision of the left eye with a + 1.50 D. sph. added was 20/40. On December 21 vision of this eye with a + 2.50 D. sph. ⊃ + 1.25 cyl., ax. 90 was 20/40.

Physical Examination.—General physical examination made at the Massachusetts General Hospital, Jan. 21, 1936, gave negative results except for numerous small telangiectases of the skin and slight hypochromic anemia. The blood pressure was 160 systolic and 80 diastolic. The red blood cells numbered 4,390,000. The Hinton test was negative. Roentgen examination of the teeth and chest gave negative results. The Graham test of the gallbladder was negative. The tonsils and urine were normal. At one time a soft mass was felt in the abdomen, but this disappeared in a few days.

Course and Outcome.—On April 3 vision of the left eye with correction was 20/70. The retinal lesion had changed in appearance. Beneath the elevated retina a dark mass could be indistinctly seen. There were no retinal hemorrhages. The vitreous was clear. A diagnosis of probable sarcoma of the choroid was made. On April 10 vision of the left eye was 20/200. The eye was enucleated.

Pathologic Examination.—Fixation, embedding and staining were carried out as in cases 1 and 2. Serial horizontal sections showing the lesion in the fundus were made.

Macroscopic Observations: On opening the eye there was seen an elevated irregular area in the macular region about twice the area of the disk. There were no hemorrhages around it. The surface was uneven, possibly due to the fixation. The yellow color of the macula was still well marked.

Microscopic Observations: On microscopic examination the eye was seen to be normal except for the lesion in the macula. Peripheral cystoid degeneration of the retina was slight below, extending backward from the ora serrata only about 1.5 mm., but it was excessive above, extending here for a distance of more than 6 mm. behind the equator. At the equator it was most marked, and almost split the retina into two thin layers. The optic disk showed an unusually deep physiologic cup. The retinal, choroidal and posterior ciliary vessels were notably free from endovasculitis. The elevation of the retina in the macular region was seen to be due to a subretinal exudate of serum. About half of this was confined beneath the pigment epithelium, which was lifted up from Bruch's

membrane in the form of a flattened intact vesicle (fig. 7). The vesicle was somewhat eccentric with reference to the fovea. Over its central portion the pigment epithelium was still adherent to the retinal neuroepithelium, but elsewhere it was separated from it by serum. The vesicle was 2.2 mm. in its greatest width and 0.35 mm. in its greatest height. Outside of the vesicle there was more serum below than above. Just below the vesicle the retina was sharply elevated 0.8 mm. in an area 3 mm. wide, but the serum extended under the retina for a total distance of 7 mm. Outside the vesicle there were no blood corpuscles or fibrin. The upper half of the vesicle was also free from these elements, but in the lower half there were two globular masses of blood corpuscles, which in a few sections took up about one-half the area of the vesicle (fig. 8). There was here also a slight amount of delicate fibrin in the serum. The pigment epithelium forming the wall of the vesicle was generally only one cell in thickness, but here and there the thickness had been increased to that of two cells. It showed no break anywhere. Many of the cells were greatly distended with serum and projected toward the retina, indicating that serum had been exuded from within the vesicle through the epithelium. At the nasal side of the vesicle there was a layer of new-formed connective tissue extending over Bruch's membrane for about 1 mm. (fig. 9). The serial sections showed that it extended vertically also about 1 mm. Beyond the margin of the vesicle it extended between the pigment layer and Bruch's membrane, gradually diminishing in thickness and disappearing at a distance of about 0.5 mm. from the margin of the vesicle. It reached its greatest thickness, about 0.4 mm., within the vesicle, where it was not in contact with the pigment epithelium. The stroma of the new tissue was hyaline and extremely delicate. It was fairly rich in fibroblasts, which were most abundant at and near its surface toward the cystic cavity. It contained a few vessels, some of them slightly larger than capillaries, but no more than two in any one section. Bruch's membrane was intact except for two small microscopic breaks. Actively proliferating fibroblasts could be seen extending from within the choroid through the breaks, and a capillary was noted passing through one of the breaks. In a few other places fibroblasts could be seen penetrating through Bruch's membrane into the new tissue without producing definite breaks in the membrane. The choroid beneath the new tissue was otherwise normal, except that it showed a few lymphocytes along the walls of some of the veins and possibly slight distention of the veins. The retina proper was slightly distorted over the vesicle but otherwise appeared perfectly normal.

COMMENT

The ophthalmoscopic picture seen in case 1 and that seen early in case 2 were essentially identical and were similar to those described in the literature as occurring early in cases of disciform degeneration of the macula. Except for the two areas of retinal opacity shown at the periphery of the mound, color plate 1 depicts the lesion in case 1 almost as accurately as it depicts that in case 2. In case 2, in which the eye was not removed until three months later, the ophthalmoscopic picture was observed gradually to change into that typical of the later stage of this disease (color plate 2).

On microscopic examination in case 1 there was observed a rupture of the choriocapillaris and, extending from this, a large hemorrhagic extravasate which had separated the pigment epithelium from Bruch's

membrane in the form of a large vesicle (fig. 1). That such a condition could occur has apparently never hitherto been demonstrated or even considered possible. It is interesting that the capillary rupture (fig. 2) was not beneath the center of the vesicle but at the periphery of the latter and hence at the periphery of the macula.

In case 2 the histologic picture was essentially the same as that seen by many other observers, with the important exception that in this case masses of blood undergoing organization were still present and thus furnished conclusive proof that the new tissue resulted from organization of blood. It was clearly evident that this blood was originally largely confined beneath the pigment epithelium, as in case 1, but its exact source could not be located, owing, no doubt, to the later stage at which the eye was removed. In this case, therefore, the possibility cannot be excluded that the blood was derived from the choriocapillaris by diapedesis. Since, as just pointed out, in cases 1 and 2 the ophthalmoscopic appearances were essentially the same as those described in the literature as occurring at corresponding stages and since in case 2 both the ophthalmoscopic and microscopic observations were essentially the same as those described in later stages of the disease, it is reasonable to conclude that hemorrhage beneath the pigment epithelium is the usual cause of senile disciform degeneration of the macula.

In case 3 there was seen a condition apparently not hitherto observed microscopically or ever recognized ophthalmoscopically, namely, a serous exudate occurring primarily beneath the pigment epithelium behind the macula and elevating the retina here in the form of a mound (figs. 7 and 8). From the standpoint of pathogenesis, the condition in this case was evidently closely related to that in the other two cases, but it is unlikely that the process would have finally resulted in the clinical picture of senile disciform degeneration. It is true that at the time the eye was removed there was definite proliferation of connective tissue on the surface of Bruch's membrane within the vesicle, but it is improbable that this would have gone on to the formation of a mass of connective tissue ophthalmoscopically conspicuous.

Clinically, also, the condition in case 3 differed from that in the other two cases in two important respects, namely, in the ophthalmoscopic picture and in the visual impairment. At the outset, in the macula there was a moundlike elevation darker than the surrounding retina but much lighter than that seen early in the other two cases. At this stage visual acuity was only slightly impaired in case 3, whereas in cases 1 and 2 central vision was almost, if not completely, abolished. The preservation of central vision for a time in case 3 was no doubt due to the fact that the pigment epithelium remained in contact with the rods and cones. Later, as was revealed by the microscopic examina-

tion, serum exuded through the epithelium and separated it from the rods and cones, thus causing loss of visual function here. At this stage there was seen with the ophthalmoscope what appeared to be a darker mass beneath the retina. This, no doubt, was the pigment epithelium separated from the retina proper, but it was mistaken for a sarcoma and led to removal of the eye. The early loss of central vision in cases 1 and 2 was probably due to the rapid and forcible elevation of the macula by hemorrhage and to the resulting distortion. It is noteworthy, however, that in each of these cases the pigment epithelium remained in contact with the fovea, although it was separated to some extent from the retina elsewhere beneath the macula by blood which had broken through at the periphery.

Owing to the age of the patient, 63 years, the condition in case 3 would not ordinarily be regarded as belonging to the juvenile type of disciform degeneration of the macula. It is not unreasonable to suppose, however, that in any patient free from arteriosclerosis a macular lesion could occur identical with that occurring in young persons. Certainly the microscopic characteristics of the lesion in this case seem to accord with the ophthalmoscopic appearances of the lesions in the juvenile type of disciform degeneration and also well explain the restoration of vision, with or without residual macular changes, that occurs in this disorder. For it is obvious that if in case 3 the serous exudate had become absorbed and the slight formation of connective tissue had ceased, visual acuity would have been largely, if not completely, restored. Moreover, the long preservation of central vision in case 3 and the absence of hemorrhage in the early stage are characteristic of the juvenile, not of the senile, type. Perhaps there will be found occurring at various ages, instances in, which the character of the lesion is intermediate between that of the senile and that of the juvenile type.

Another condition that must be considered in connection with case 3 is so-called serous retinitis. This is often central; in fact, it is only the central type that one of us (F. H. V.) has ever observed. In such cases a large central area of the retina, including the macula but often extending beyond it, becomes diffusely cloudy and may show a wavy appearance resembling ripples of sand on the seashore. Vision is markedly impaired at the onset, but the condition always subsides, with recovery of good vision. The cause of this disorder has never been determined, but the observations in case 3 suggest that it may be serous exudation from the choroid. In serous retinitis, perhaps, the serum is under less pressure, is slowly exuded through the pigment epithelium without lifting it up and then permeates beneath or into the retina.

From a histologic standpoint, our three patients showed several features of considerable additional interest. In case 1 Bruch's mem-

brane showed only one break, a small one at the site of the rupture of the choriocapillaris, from which the hemorrhage arose. In case 2 there were noted only a few breaks in Bruch's membrane. These were very small, and through two of them new vessels passed from the choroid into the mound (figs. 5 and 6). The other breaks were filled with fibroblasts. Evidently, however, most of the connective tissue cells in the mound were derived from cells which had penetrated Bruch's membrane without producing microscopically visible breaks.

In case 3 only a very few small capillaries were observed extending through Bruch's membrane. In this case the proliferation of connective tissue cells through Bruch's membrane without causing breaks could be definitely recognized, for small spindle-shaped accumulations of such cells were seen here and there on the inner surface of the membrane, and occasionally a cell was observed extending through the membrane. In general, there were no accumulations of cells beneath the membrane within the choroid, although in a few instances slight accumulations were seen. The largest of these comprised about thirty cells in the section. No evidence could be observed that the retinal vessels played any part in the process of organization in either case 1 or case 3. In case 3 it was evident that serum was transudating from within the vesicle through the pigment epithelium. The latter showed no breaks, even microscopically, but many of its cells were distended with serum, which often could be seen to have broken from the cells into the space beneath the retina proper. A noteworthy feature in case 2 was the absence of hematogenous pigment in spite of the large amount of blood still remaining in the tissue. In case 2 it was clearly evident that the pigment epithelium played an important part in the process of organization. For from the epithelium large strands of cells were observed extending into masses of blood, and all stages in the formation from pigment cells of cells free from pigment and indistinguishable from fibroblasts were observed. In other places also the origin of the cells of the stroma from pigment epithelium was evident. In many places, however, it could not be determined whether the cells were of epithelial or mesoblastic origin, although no doubt those closely associated with the blood vessels were mesoblastic. In case 3 the new tissue was almost, if not entirely, mesoblastic in origin. What caused its formation in this case is not clear. There was no blood in its vicinity at the stage at which microscopic examination was made, but possibly blood had previously been present here. There was no condition seen in the choroid to explain its formation.

A question that arises in connection with the histogenesis of these lesions is whether similar lesions do not occur elsewhere than beneath the macula. One of us (F. H. V.) has examined sections of a number of eyes in which there was extensive formation of connective tissue between

the retina and Bruch's membrane elsewhere than in the macula, tissue similar to that seen in the advanced stage of disciform degeneration, but, unfortunately, has no evidence as to its histogenesis. Probably such conditions would not be recognized clinically as analogous to disciform degeneration of the macula.

Another question is whether the condition is related to Coats' disease. One of us (F. H. V.) has had an opportunity to examine sections of the lesions in the latter disease in both the early and the late stages and has found that the new tissue is formed in the external part of the retina but not primarily beneath the pigment epithelium. In the early stages there is a large amount of fibrin and hematogenous pigment present. In the later stages, cholesterol crystals surrounded by foreign giant cells may be numerous. It would seem, therefore, that Coats' disease is essentially different from disciform degeneration of the macula. The former disease seems to be primarily retinal, the latter choroidal, in origin.

In cases 1 and 2, hemorrhagic extravasates extended beneath the retina from the periphery of the mass. The microscopic examination in case 1 showed that such extravasates resulted from blood breaking through the pigment epithelium at one or more places at the periphery. In case 2 there were seen ophthalmoscopically small hemorrhagic extravasates apparently not connected with the main mass. These were noted to be entirely subretinal, so that actually they must have been originally derived from the main mass of blood. No doubt many of the separate retinal hemorrhages depicted in recorded cases also had this origin. In case 2 the gray retinal opacities below the mass and above the optic disk were observed to be due to serum beneath the retina. This serum contained a few discrete collections of red blood cells. Since the retina showed no edema or hemorrhage, it is evident that this serum had separated from the main mass of blood, just as serum usually does from any collection of stagnant blood. Probably some of the peculiar appearances depicted in various recorded cases can be explained in this way.

The causes of the hemorrhages in case 1 and 2 and of the serous exudate in case 3 remain to be determined. Behr has advanced the view that disciform degeneration of the macula is due to some alteration in Bruch's membrane which permits an exudate to occur beneath the retina. But to us this seems highly improbable. It is true that with advancing age certain changes occur in the elastic portion of Bruch's membrane, as was first pointed out by Verhoeff and Sisson, but these changes are not more marked in the macular region than elsewhere, and even when very marked they are not necessarily associated with any alteration in the retina or impairment of vision. The inner portion of the membrane may also undergo senile change such as is commonly

seen associated with colloid excrescences, but this also does not lead to exudation from the choroid. Behr stated that in his case Bruch's membrane was split into two layers at the margin of the lesion, but it seems to us more likely that a new cuticular membrane had been formed by the proliferating pigment epithelium here. In advanced cases such as his it seems remarkable to us not that Bruch's membrane was altered beneath the mass but that it was so slightly altered.

The observations in our cases clearly indicate that disciform degeneration is due to some disturbance, possibly only temporary, in the choriocapillaris of the macular region, since they show the primary condition to be hemorrhage or serous exudation beneath the pigment epithelium. Obviously, the cause of the vascular disturbance in the choroid could be the same or different in different cases. The fact that the disorder most commonly occurs in patients of advanced age suggests that general angiosclerosis is a predisposing factor or that some condition associated with angiosclerosis is often an important factor in producing changes in the choriocapillaris. However, the observations in our cases, as well as those in the cases recorded in the literature, afford no support for the view that general angiosclerosis is always the cause of the disorder. For in the recorded cases sclerosis of the retinal vessels has been mentioned as occurring in only twenty-five of one hundred and twenty-nine eyes, and in our cases there was no evidence of angiosclerosis, either local or general, aside from insignificant sclerosis of some of the choroidal vessels in case 2. On the other hand, the observations in our cases do not exclude the possibility of a localized vascular lesion of infectious or other origin, since obstruction of a small vessel in the affected region could have escaped observation in spite of serial sections. In none of our cases did the choroid behind the lesion show recognizable edema, congestion or, aside from the site of the rupture in case 1, hemorrhage. Edema and congestion, however, could have disappeared before the eyes were removed if the vascular disturbance had been compensated or had subsided.

The fact that disciform degeneration of the macula has been observed in cases of angioid streaks is in accord with the conclusions at which we have arrived. For choroidal hemorrhages are frequent in cases of the latter condition and indicative of vascular disturbances which might well lead to hemorrhage or serous exudation from the choriocapillaris behind the macula.

There are a considerable number of cases in which disciform degeneration of the macula and circinate retinitis coexist. These comprise a high percentage of cases of the former condition, but a low percentage of cases of the latter. It seems evident that the two conditions must have at least one important cause in common or that one causes the other in these cases. The lesions in circinate retinitis appear to be

entirely retinal and consist chiefly of deposits of lipoid substance within large spaces in the retina about the macula and the phagocytosis of this substance by macrophages. There are also often small retinal hemorrhages. Ophthalmoscopically recognizable sclerosis of the retinal vessels may be present but is often absent. There is no known systemic metabolic disturbance to explain the condition. Since lack of oxygen is a cause of deposition of fat in the tissues, it seems possible that disturbance in the capillary circulation of the macular region may be the chief cause of circinate retinitis, as it probably is of the retinal hemorrhages often observed in these cases.

The macula is a specially differentiated vascular bed, as is also the choriocapillaris behind the macula. Hence it seems likely that when capillaries of the choriocapillaris are affected in some special way, those of the macular region of the retina are apt to be similarly affected. For the same reason also, it would be expected that each eye would often be similarly affected, a conclusion that accords with the fact that disciform degeneration and circinate retinitis are often bilateral. Since the structure of the retina is entirely different from that of the choroid, similar vascular changes in them might well produce different effects, in the one hemorrhages and deposits of fat, and in the other transudation of serum or hemorrhage by diapedesis or rupture. The fact that only rarely have disciform degeneration and circinate retinitis coexisted in opposite eyes can be explained by assuming that when alteration of the choroidal vessels is more marked in one eye, alteration of the retinal vessels is apt to be more marked in this eye than in the other.

On the other hand, the possibility cannot be dismissed that in at least some of the cases in which circinate retinitis and disciform degeneration coexist in the same eye, the former has predisposed to, or -actually caused, the latter. For in advanced cases of circinate retinitis the pigment epithelium shows marked changes, and it thus seems possible that the same deleterious influence which has produced these changes may cause hemorrhage or serous exudation from the choriocapillaris.

The foregoing reasoning suggests the possibility that senile pigmentary changes in the macula may predispose to disciform degeneration, but this possibility seems remote, because such senile changes may be extremely marked without leading to hemorrhage from the choriocapillaris. It is, however, in accord with the facts that disciform degeneration in one eye is sometimes preceded by pigmentary macular changes in the same eye and is often associated with such changes in the other eye.

In arriving at a diagnosis of disciform degeneration of the macula the chief considerations are the size and situation of the elevated mass, the texture of its surface, the absence of inflammatory signs, the absence

of extensive separation of the retina, the coexistence of circinate retinitis and the presence of subretinal hemorrhagic extravasates extending out from the periphery of the mass. Such extravasates existed in our cases 1 and 2, have been described in many of the reports of the previously recorded cases and are therefore of great diagnostic importance when present. In Coats' disease the patient is almost always younger and the lesion larger than in any case of disciform degeneration. If, however, as might conceivably happen, the lesion in Coats' disease was small and confined to the macula, it could not be certainly distinguished ophthalmoscopically from disciform degeneration. It is more important to distinguish the latter from malignant melanoma and thus avoid needless removal of the eye. A small malignant melanoma situated in the macula and closely resembling disciform degeneration seldom occurs. In only one instance in the Massachusetts Eye and Ear Infirmary has such a tumor been mistaken for disciform degeneration. In this case the tumor showed several hemorrhagic extravasates due, as was afterward determined by microscopic examination, to necrosis of the tumor. Although situated near the periphery, they did not extend from beneath the mass. The mistake in diagnosis was soon rectified and did not delay removal of the eye more than two months. Much more frequent is the mistaking of senile disciform degeneration for malignant melanoma. This mistake was made by the senior writer in case 1. At that time it was unknown that a hemorrhage beneath the pigment epithelium could occur and produce the appearance of a perfectly black mound in the macula. The experience afforded by this case enabled one of us (F. H. V.) immediately to make a correct diagnosis five years later, when early in the course in case 2 an almost identical ophthalmoscopic picture was seen. In case 3 a diagnosis of disciform degeneration was made at first and was later abandoned when the appearance of the lesion changed. This mistake probably could have been rectified if enucleation had been delayed longer. We now believe that a malignant melanoma so seldom ophthalmoscopically presents the appearance of disciform degeneration that in a case of suspected tumor removal of the affected eye should be delayed until no suspicion remains that the lesion is that of disciform degeneration of the macula.

SUMMARY AND CONCLUSIONS

The literature relating to disciform degeneration of the macula is reviewed, and eighty-four cases of the senile type, one hundred and twenty-nine eyes being affected, are analyzed.

The clinical, ophthalmoscopic and microscopic observations in three cases of macular disease are described.

The observations in two of these cases prove conclusively that a hemorrhagic extravasate can occur between the pigment epithelium and Bruch's membrane, undergo organization and produce an ophthalmoscopic picture typical of senile disciform degeneration of the macula.

That this is the usual, if not the only, pathogenesis of this disease is strongly indicated by a comparison of the observations in these cases with those of the cases recorded in the literature.

In the third case the microscopic examination revealed the pigment epithelium lifted up in the form of a large vesicle by serous exudate. It is suggested that such a serous exudate may be the cause of the juvenile type of disciform degeneration of the macula and that so-called serous retinitis may have a similar origin.

The extravasation of blood or serum beneath the pigment epithelium in disciform degeneration of the macula is due to some disturbance in the choriocapillaris. Since the disorder is most commonly a senile disease, localized angiosclerosis is probably the usual cause of this vascular disturbance. In certain cases, especially those of the juvenile type, the vascular disturbance may have been dependent on some other cause or causes yet to be determined.

The frequent association of disciform degeneration of the macula with circinate retinitis may be due to the fact that each occurs in specially differentiated vascular beds. The possibility cannot be excluded, however, that in some cases, at least, circinate retinitis causes disciform degeneration by producing hemorrhage or serous exudation from the choriocapillaris.

In a doubtful case of malignant melanoma of the choroid, removal of the affected eye should be delayed until no suspicion remains that the lesion is that of disciform degeneration of the macula.

BIBLIOGRAPHY

Pagenstecher, H., and Genth, C. P.: Atlas der pathologischen Anatomie des Augapfels, Wiesbaden, C. W. Kreidel, 1875; cited by Junius and Kuhnt.

Michel, J.: Ueber Geschwülste des Uvealtractus, Arch. f. Ophth. **24**:131, 1878.

Walker, C. H.: A Case of New-Growth in the Macular Region, Tr. Ophth. Soc. U. Kingdom **17**:4, 1897.

Batten, R.: Peculiar Symmetrical Swellings in the Macular Region Apparently Due to Subretinal Hemorrhage, Tr. Ophth. Soc. U. Kingdom **24**:127, 1904.

Possek: Ueber senile Macularveränderung bei Arteriosclerose, Ztschr. f. Augenh. **13**:771, 1905.

Oeller, J.: Atlas seltener ophthalmoscopischer Befunde, Wiesbaden, J. F. Bergmann, 1905; cited by Julius and Kuhnt.

Lawford, J. B.: A Case of Subretinal New Growth of Doubtful Nature, Tr. Ophth. Soc. U. Kingdom **31**:257, 1911.

Axenfeld, T.: Retinitis externa exsudativa mit Knochenbildung im sehfähigen Auge, Arch. f. Ophth. **90**:452, 1915.

Hegner: Retinitis exsudativa bei Lymphogranulomatosis, Klin. Monatsbl. f. Augenh. **57**:27, 1916.

Beatson, H.: Notes on a Case of Tumor in the Retina (Massive Exudation), Tr. Ophth. Soc. U. Kingdom **36**:345, 1916.

Holm, E.: Retinitis exsudativa externa, Klin. Monatsbl. f. Augenh. **59**:319, 1917.

Elschnig, A.: Tumorähnliche Gewebswucherung in der Macula lutea, Klin. Monatsbl. f. Augenh. **62**:145, 1919.

Knapp, A.: Subretinal Exudate Simulating Sarcoma of the Choroid with Anatomic Examination, Arch. Ophth. **48**:559, 1919.

Neame, Humphreys: A Case of Retinitis Circinata, Proc. Roy. Soc. Med. **16**:11, 1923.

Coppez, H., and Danis, M.: Rétinite exudative maculaire senile, Arch. d'opht. **40**:129, 1923.

Feingold, M.: Senile Exudative Macular Retinitis with Remarks on Similar Tumor Formation in a Case of Angioid Streaks, Tr. Am. Ophth. Soc. **22**: 268, 1924.

Cords: Scheibenförmige Maculadegeneration, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **45**:246, 1925.

Junius, P., and Kuhnt, H.: Die scheibenförmige Entartung der Netzhautmitte, Berlin, S. Karger, 1926.

Verhoeff, F. H., and Sisson, R. J.: Basophilic Staining of Bruch's Membrane, Arch. Ophth. **55**:125, 1926.

Ormond, Arthur W.: Four Cases of Disc-Like Degeneration of the Macula Lutea, Guy's Hosp. Rep. **7**:16, 1927.

Paul, L.: Choroiditis exsudativa unter dem Bilde der scheibenförmigen Entartung der Netzhautmitte, Ztschr. f. Augenh. **63**:205, 1927.

Cushman, B.: Disciform Degeneration of the Macula, Am. J. Ophth. **11**:720, 1928.

Weizenblatt, S.: Fall von scheibenförmiger Netzhautdegeneration bei 40-jährigem Patienten, Ztschr. f. Augenh. **65**:299, 1928.

Holloway, T. B., and Verhoeff, F. H.: Disc-Like Degeneration of the Macula, Tr. Am. Ophth Soc. **26**:206, 1928.

Barriére, V. A.: Disciform Degeneration of Central Region of the Retina, Arch. de oftal. de Buenos Aires **4**:223, 1929.

Junius, Paul: Erscheinungsformen und Ablauf der juvenilen Retinitis exsudativa macularis, Ztschr. f. Augenh. **70**:129, 1929.

Hanssen, R.: Zur Anatomie der scheibenförmigen Degeneration der Netzhautmitte, Ztschr. f. Augenh. **72**:360, 1930.

Pallarés, Juan: Degeneratio disciformis maculae luteae, Klin. Monatsbl. f. Augenh. **86**:201, 1931.

Behr, Carl: Ein weiterer Beitrag zur Anatomie und zur Pathogenese der scheibenförmigen Degeneration am hinteren Augenpol, Ztschr. f. Augenh. **75**:216, 1931.

Duynstee, H. P. M.: Degeneratio maculae luteae disciformis, Klin. Monatsbl. f. Augenh. **88**:511, 1932.

Adler, F. H.: Disciform Degeneration of the Macula (Junius and Kuhnt), Arch. Ophth. **10**:379 (Sept.) 1933.

Kahler, A. R., and O'Brien, C. S.: Disciform Degeneration of the Macula, Arch. Ophth. **13**:937 (June) 1935.

Davis, W. T., and Sheppard, E.: Juvenile Macular Exudative Retinitis (Junius), Arch. Ophth. **13**:960 (June) 1935.

LOCALIZATION OF CHANGES IN THE EYEGROUND AND PEILINGATION OF THEIR PROJECTIONS ON THE SCLEROTIC

REPORT OF A NEW METHOD

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The precise localization of pathologic changes or a foreign body in the eyeground is one of the important and difficult problems of ophthalmology.

At the Thirteenth International Congress of Ophthalmology (held in Amsterdam in 1929), in discussing the operative treatment of retinal detachment, Lindner, Igersheimer, Brückner, Löwenstein and others pointed out that the precise localization of retinal rupture and the touching of this place with a thermocautery are very difficult and that the operation for detachment of the retina cannot become popular among ophthalmologists so long as new methods are lacking for the localization of the ruptured place and for the finding of its projection on the sclerotic when operating.

It is equally important to be able to localize precisely a foreign body adjacent to the eyeground, particularly in the posterior section of the eyeball.

Professor Natanson found search for a foreign body in the vitreous with a magnet to be harmful, as the posterior shriveling of the vitreous may involve detachment of the retina. He pointed out that retinal detachment after operation will occur much less frequently if localization of the foreign body is made with more precision. This pertains both to roentgenologic localization and to localization of the foreign body near the membrane of the eye by means of the ophthalmoscope. The precise localization of such an intra-ocular body may permit its extraction diasclerally with a magnet, almost without involving the vitreous and without unnecessary traumatization of the neighboring retinal parts; this may greatly diminish the danger of the occurrence of retinal detachment in the future. There exists a relatively extensive literature on the localization of intra-ocular cysticerci, initiated by Alfred von Graefe. If it is remembered, in addition, that a tumor or an inflammatory focus in the eyeground which is seen ophthalmoscopically is not measured in

clinical practice (in my opinion, only because sufficiently elaborated methods do not exist for such measurement and localization which are accessible for every ophthalmologist), it will be evident that it is necessary to bring clearness and precision to the study of the localization of an intra-ocular change (such as a retinal rupture or an inflammatory or atrophic focus) or of a foreign body or a cysticercus near the membrane of the eye.

The study of such changes in the eyeground consists of two parts, which are equally important for the ophthalmologist. The first part treats of the precise localization of the morbid focus in the eyeball with reference to definite points of the eye, in either of the coordinates, which can be expressed in degrees or in linear measures. The object of the second part is the study of the best methods of topographic localization on the sclerotic of the point where it is necessary to make an incision or a cauterization when operating. The immediate finding of these points on the sclerotic is called peilingation, the term used by Tzeekoolenko. In a third part a new method of localization and peilingation will be discussed.

In the first part of the study of the localization my aim is to define the coordinates of the morbid focus in the eyeball. These coordinates must be given both for the surface of the eyeground and for the exterior surface of the eye.

I. LOCALIZATION OF THE MORBID FOCUS IN THE EYEBALL

Methods of Definition of a Scotoma Corresponding to the Morbid Focus in the Eyeground.—A disadvantage of these methods is the impossibility of applying perimetry and campimetry in cases in which the affected eye has poor visual acuity. Besides, the dimensions of a scotoma correspond, for the most part, to a much larger area of the retina than that of the morbid focus. Donders was the first to suggest that such scotomas be examined. He devised a ready reckoner for the defining of the distance from the limbus of the affected part of the eye. But this table is far from complete, is insufficiently precise and has become obsolete.

Donders examined a scotoma, i.e., he defined its location, by means of the general perimetric method. Goldfeder suggested that the location of a scotoma be defined by means of fire point perimetry in the dark. For this purpose he used an electric ophthalmoscope as an object, placed in front of it a diaphragm with a small opening and moved the ophthalmoscope along the arc of the perimeter.

Haase defined the location of a scotoma with a campimeter. But, owing to the errors in his calculations, this method cannot be regarded as an accurate one.

Methods of Measurement of the Distances in the Eyeground in Conformance with the Diameter of the Disk (Papilla Diameter).—To this group of methods belong those evolved by Schmidt-Rimpler, Kleefeld and Gonin. They are based on the discovery, by the judgment of the eye, of how many diameters of the optic disk could be set in a row along a certain distance. Therefore, their exactness depends largely on the ophthalmologist's judgment by the eye, and the individual error may sometimes be considerable. According to Schmidt-Rimpler, the diameter of the optic disk is admitted to be 1.5 mm.; the distance of the temporal margin of the optic disk from the posterior pole of the eye, 3.5 mm.; the radius of the eyeball, 12 mm.; its circumference, 74 mm., and the diameter of the cornea, 12 mm. Therefore, the distance from the margin of the cornea to the posterior pole must be 37 mm. less 6 mm., or 31 mm.; the distance between the margin of the cornea and the margin of the optic disk along the temporal side is 34.5 mm., and that along the nasal side, 26 mm.

Of course, Schmidt-Rimpler's calculations are approximate, but one cannot obtain greater accuracy. The diameter of the optic disk is conditionally admitted to be 1.5 mm., but the anatomic measurements of many authors show that the optic disk may vary in diameter from 1.2 to 1.8 mm.

For better orientation when measuring the eyeground Kleefeld suggested that a mesh be placed on the magnifying glass and the divisions of this mesh be compared with the size of the optic disk.

For the localization of retinal ruptures Gonin suggested his own method, based on the same principle. When during examination in a passable light Gonin found that the retinal rupture flashed as a red light on the background of the detachment, he marked at the limbus with india ink the meridian he had found. Then he measured the distance from the ora serrata in papilla diameters. If the ora serrata cannot be viewed, the distance in papilla diameters is measured from the exterior point on the periphery, which is visible with the ophthalmoscope. Then the number of papilla diameters is multiplied by 1.5 mm., and to the product is added 7 mm. (for the nasal side) or 8 mm. (for the temporal side), i. e., the distance of the ora serrata from the limbus. Certainly the exactness of Gonin's method is problematic.

Methods Based on the Principle of Perimetric Localization (Ophthalmoscopic Perimetry).—All the methods of this group are based on the principle suggested by Alfred von Graefe. The angle lying between the optic line and the line drawn from the retinal morbid focus through the nodal point of the eye to the observer's eye is measured perimetrically. The incline of this line toward the horizontal plane is also reckoned. For this purpose Graefe used a special ophthalmoscope.

The method which Graefe proposed in 1882 for the localization of a cysticercus was modified by Lindner and again applied for the localization of retinal ruptures (in 1929). The arc of the perimeter is attached to a large Gullstrand electrical ophthalmoscope, and in its ocular are inserted crossed threads. Along the arc of the perimeter is moved an electric lamp (for fixation of the visual line). The ophthalmoscopic image of the yellow spot coincides with the crossed threads in the ocular, and that of the retinal rupture is obtained when the eye is turned. The angle lying between two lines, the guiding line (from the cross to the retina) and the optic line (from the lamp to the yellow spot), is measured. The meridian is reckoned in conformance with the position of the arc of the perimeter.

It is obvious that if one modifies Graefe's method, placing the fixation point in the center of the perimeter and letting the observer move the ophthalmoscope along the arc of the perimeter, one may see also the extreme periphery of the eyeground. Besides, if the affected eye has reduced visual acuity, one may, by using the concomitant ocular movements, attain the fixation of the central mark of the perimeter.

All authors who applied the perimetric principle of localization followed this procedure. Dupuy-Dutemps and Pollack proposed this rule in 1910. But these authors made an error, because they considered the number of the perimetric degrees equal to the number of the arc degrees on the retina. Having defined the number of degrees of ophthalmoscopic divergence on the arc of the perimeter, they reckoned in the following manner: The divergence on the perimeter equals n degrees, and the number of the arc degrees on the retina also equals n degrees; then according to the formula $\frac{2\pi r}{360}$ n the length of the arc from the yellow spot to the morbid focus was calculated. The semicircumference of the eyeball was considered as equal to 36.5 mm. But this could be true only when the nodal point was located in the center of the eye turning. On this account the reckonings by this method are incorrect. In proportion as the focal distance from the posterior pole increases, the error of the localization becomes much greater, and the difference may attain 30 degrees·even on the equator of the eyeball.

Professor Levitzky and Professor Goldman obviated this defect in 1927. They found a relation between the visual angle and the central angle, the latter of which it is necessary to know in reckoning the length of the arc, that is, the distance between the posterior pole of the eye and the focus in the retina. These authors used Gullstrand's schematic eye as a basis for the reckonings.

But the value of this work was considerably diminished by the fact that these authors used for the dimensions of the eyeball a series of arbitrary alterations which involved a perceptible error.

However, the table by Levitzky and Goldman appears to come much nearer to the truth than any other such table.

Other authors used the same type of ophthalmoscopic perimetry (Lenz, E. G. Lasarev, Weve and Reitsch). Weve's substituting method is original. When the perimetric examination of the affected eye is performed, a model is placed on this eye constructed according to the constants of Gullstrand's schematic eye. From an electric ophthalmoscope a ray of light is projected in a formerly defined direction, and on the surface of the wall of this model appears a bright spot which indicates the location of the focus. The distance from the limbus is measured in millimeters directly on the model of the eye.

A Method for Localization of the Focus According to the Distance of the Three Points in the Field.—This method was proposed by Booshmich and Roschat independently of each other. It may be called a tangential method. The method devised by Booshmich is very simple. All the measurements are performed with the aid of two simple rulers, each 75 cm. long. During the examination these rulers are placed at right angles. One ruler is attached horizontally to a support, which is placed at a distance of 30 cm. from the eye to be examined, so that the upper verge of the ruler divides the cornea in two. The other ruler, the vertical one, is moved along the first ruler to the right and to the left. The observer is situated behind both rulers and examines the patient's eye with an ophthalmoscope. At the same time the patient's eye is fixed on a candle light or on a marking attached at the zero point of the horizontal ruler. As soon as the morbid focus in the eyeground is found by means of the ophthalmoscope, the vertical ruler is placed so that the ray of light which is projected from the ophthalmoscope to the patient's eye may fall on the upper end of this ruler. Then the readings showing in centimeters the distance of the vertical ruler from the zero point and the elevation in centimeters of its upper end above the horizontal surface are checked. Thus the distances of the three points in the field are measured: (1) that from the fixation point (30 cm.), (2) that from the point of intersection of the two rulers and (3) that from the upper end of the horizontal ruler. With the aid of rather simple calculations, based on the constants of Werbitzky's schematic eye, Booshmich found the distances of the morbid focus from the limbus and the horizontal meridian (along the chord) for the reckoning of every 5 cm. on both rulers. Thus with the aid of the Booshmich ready reckoner and two simple rulers it is possible to localize rather precisely the morbid focus.

Pavia takes photographs of the eyeground from the optic disk up to the morbid focus and with the obtained photopanorama computes the location of the morbid focus. This is a complicated method.

As soon as the morbid focus on the retina and its projection on the sclerotic are localized, that is, as soon as the coordinates on the eyeball are found by either method, the next problem is to find this point according to the coordinates of the live eye, to define exactly the place of the rupture and to perform the operation. As has already been stated, Tzeekoolenko called this preparatory determination peilingation, that is, the finding of the point according to the already ascertained coordinates.

Peilingation, according to the methods suggested up to the present, consists of two stages: (1) the marking of a point on the meridian of the eye corresponding to the place of the focus and (2) the measuring of the focal distance from the recognized anatomic point—the margin of the cornea. The first of these procedures is generally performed according to the judgment of the eye (Gonin, Odintzov, Levitzky and others). The second is performed in various ways. Gonin measured the distance from the limbus to the focus by means of a divider and put the legs of the latter along the guiding thread, which is fastened at two different points of one meridian and divides the cornea into halves. Odintzov knots the guiding thread at the limbus and then measures the necessary number of millimeters from the knot and cuts the thread there. The end of this thread shows the place of rupture. Amsler suggested that a set of special metallic indicators of various sizes be used (for measuring the distance from the limbus to the focus). Loginov and Booshmich solved this question rather differently. In the first place, the distance from the limbus along the horizontal meridian (the external or the internal rectus muscle) is measured; then from the ascertained point the distance of the focus from the horizontal meridian is measured up or down (vertically). Loginov does this by means of a silk thread on the sclerotic. Booshmich measures both distances on the chord by means of a divider. But none of these methods guarantees the precise peilingation of the focus in the eyeball.

Lindner and Tzeekoolenko suggested special instruments by the aid of which one may measure on the sclerotic the necessary number of millimeters from the limbus. Lindner suggested the use of a device made of gutta-percha paper (a ring meridian) but soon abandoned it.

Guist's instrument has a fault. The screw which fixes the arrow is located above the corneal pole; therefore, it is impossible to turn the eye too far. The eye cannot be hidden under the lids, as the instrument prevents this. This defect is present in a greater degree in Tzeekoolenko's instrument. Its vertical handle, which is placed above the cornea, is 6 cm. long. Hence it would appear that peilingation of the focus during operation on the posterior segment of the eyeball, on and behind the equator, would be difficult and sometimes impossible to perform.

II. RECKONING OF THE POINTS OF THE PROJECTION OF INTRA-OCULAR CHANGES ON THE SCLEROTIC

The precise methods of localization and peilingation must satisfy the following conditions:

1. There must be faultlessness as regards the laws of physiologic optics. 2. It is necessary to take into consideration the influence of the variations (*a*) of the size of the cornea and (*b*) of the length of the optic axis. 3. The method must be simple and must not require complicated instruments. 4. The localization and the peilingation must not require much time either before or during the operation. 5. It must be possible to apply the same method for the localization and peilingation of various changes of the eyeground, from retinal rupture to changes due to a nonmagnetic foreign body near the walls. 6. The possibility of a wider application of such a method in cases of various
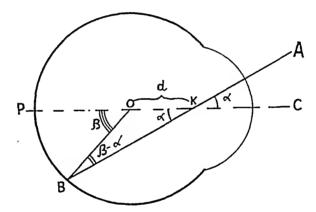
Fig. 1.—Werbitzky's reduced eye, illustrating the method of calculating the location of a morbid focus.

locations of the morbid focus in the eyeball (from the center to the extreme periphery) should be considered.

Beyond all question the method of ophthalmoscopic perimetry appears to be the most handy; hence most of the methods suggested up to the present day are based on this principle. In contrast to Lindner, who based his calculations on the eye, and to Listing, and Levitzky and Goldman, who used Gullstrand's nonprecisely schematic eye, I make all calculations according to the constants of Werbitzky's reduced eye.

If AB (fig. 1) denotes the direction of the ray of light from the examiner's eye, A to the morbid focus, B, then the angle on the perimeter between the line of fixation and the guiding line AB will equal the visual angle AKC.[1]

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1. In all further calculations it is supposed that the angle AKC is corrected on the size of the γ angle, which must be precisely defined on the patient before starting the ophthalmoscopic perimetric study.

For the definition of the location of the focus, B, as regards the posterior pole of the eye, P, it is necessary to know the central angle β (POB). It is exterior to the triangle BOK and equals the sum of the two proximate angles, that is OKB and OBK. The angle OKB equals the visual angle a. The angle OBK (or the angle $\beta - a$) can be easily reckoned from the oblique-angled rectilinear triangle OBK according to the well known axiom: The sides of a triangle coordinate as the sides of the opposite angles. If OK $= d$, i. e., the distance of the nodal point from the centrum, and OB $= r$,

$$\frac{d}{r} = \frac{Sin\ (\beta - a)}{Sin\ a}$$

$$\frac{d}{r} \cdot Sin\ a = Sin\ (\beta - a).$$

This is the basic formula for the calculations. According to the constants of Werbitzky's reduced eye, d is 6.4 mm., and the radius of the retinal curvature, r, is 10.2 mm. The equation then becomes:

$$\frac{6.4}{10.2} \cdot Sin\ a = Sin\ (\beta - a).$$

After the reckoning of this equation with the aid of mathematical tables the angle $(\beta - a)$ is defined. By adding it to the angle a, the central angle β is obtained. During the further localization of the focus B, one may proceed in two ways. One may reckon on the sclerotic the distance (in millimeters) of the focus from the limbus, or one may define the distance of the focus from the anterior pole. The first mode is common to all methods without exception, and the second is the basis of the new method of localization and peilingation suggested now by me.

I shall discuss the first mode. One of the essential defects in reckoning the distance in millimeters is, to my mind, the lack of consideration of the variations in the corneal diameter. To define the influence of these variations I study the triangle ODL (fig. 2). The line DL represents one half of the diameter, and OL, the radius of the principal part of the eyeball from the centrum to the surface of the sclerotic. If it is admitted that the wall of the eye measures 1 mm. (the retina measures 0.4 mm.; the vascular membrane, 0.04 mm., and the sclerotic, 0.5 mm.), I assume that OL equals 11.2 mm. (10.2 + 1.0 mm). For the various possible corneal dimensions from 9.5 to 12.5 mm. (already mentioned), DL accordingly equals from 4.25 to 6.25 mm. From the rectangular triangle ODL is reckoned the correlation $\frac{DL}{OL} = Sin\ \delta$; for instance, $\frac{4.25}{11.2} = Sin\ \delta$, etc. With the mathematical tables one finds that the dimension may vary from 25.1 to 33.9 degrees.

These data are obtained if one admits that side by side with the wide variations of the corneal diameter the length of the radius of the eyeball (11.2 mm.) remains unalterable. But there is a strong pre-

sumption against this. Just as an average radius for the curvature of the surface of the eyeball was assumed, an average angle must also be chosen; that is, an average corneal diameter must be taken for the calculations. According to my measurements on six hundred eyes, the average length of the horizontal corneal diameter equals 11.13 mm. and that of the vertical diameter 10.36 mm. The arithmetical mean is 10.74 mm. As the average radius of the surface of the sclerotic (according to the constants of the reduced eye) I use the average size of the corneal diameter which I obtained with my measurements. Thus, in accordance with the equation $\frac{5.3}{11.2} = \mathrm{Sin}\ \delta$, the angle δ equals 28.65 degrees. Next in order is the reckoning of the length of the arc B_1L, i. e., the distance of the projection of the focus on the sclerotic (B_1) from the corneal border. The arc B_1L is drawn together by the central angle ϵ, which equals 180 degrees $-(\beta - a)$. I calculated the angle β after defining
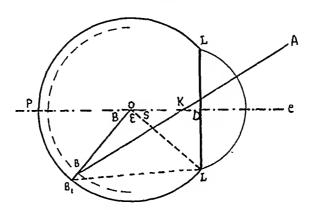
Fig. 2.—Reduced eye, illustrating the method of reckoning the angle δ.

the visual angle L; the size of the angle δ was obtained after measuring the corneal diameter.

When the size of the angle ϵ is known, one can reckon in millimeters the length of the arc B_1L in accordance with the formula $\frac{\pi K \epsilon}{180}$. Taking these calculations as a basis, I reckoned in millimeters a table of distances of the projection of the focus from the limbus in conformity with the size of the visual angle for every 5 degrees (table 1).

It must be borne in mind that all these calculations were made on the assumption that the length of the axis of the eyeball and the radius of its surface remain constant. But it is well known that they often vary. The lengthening of the diameter of the posterior half of the eyeball has a particular meaning in myopia. Lindner pointed this out and in cases of lengthening of the optic axis (in myopia) proposed to add 1 or 2 mm. to the reckoned distance from the limbus. However, this lowers the exactitude of the peilingation.

As for the variations in the radius of the surface of the eyeball, it is evident that every considerable alteration of its length involves considerable lengthening or shortening of the surface of the eyeball. In such cases the readings in millimeters on the sclerotic cease to be correct, and table 1, which shows precise reckonings only for the radius of 11.2 mm., loses its value. These data will be erroneous in cases of more or less considerable anatomic variations of the eyeball. In order to lessen, so far as possible, the influence of this factor on the exact solution of the problem under consideration, I elaborated a new method of localization and peilingation.

TABLE 1.—*Calculations in Millimeters of the Position of the Morbid Focus on the Sclerotic*

Visual Angle	Distance From the Posterior Pole of the Eye	Distance From the Limbus
5	1.6	28.0
10	3.2	26.1
15	4.8	24.8
20	6.3	23.3
25	7.9	21.7
30	9.4	20.2
35	11.0	18.6
40	12.5	17.1
45	13.9	15.7
50	15.4	14.2
55	16.8	12.8
60	18.2	11.4
65	19.5	10.1
70	20.7	8.9
75	22.0	7.6
80	23.1	6.5
85	24.2	5.4
90	25.2	4.4

III. NEW METHOD OF LOCALIZATION AND PEILINGATION

This method is very simple. The two coordinates of the projection of the focus on the sclerotic are denoted in degrees. Until recently all authors measured only one coordinate in degrees (the angle between the meridian on which the focus is located and the horizontal meridian), and the other coordinate was denoted in linear measures (millimeters), because it measured on the sclerotic the distance of the focus from the limbus. I denote the second coordinate in degrees as the distance of the focus from the anterior pole of the eye. Hence my method does not require many calculations. I determine by means of ophthalmoscopic perimetry what central angles (the distance in degrees of the focus from the posterior pole of the eyeball) correspond to the visual angles. The obtained angle, β, is deducted from 180 degrees, and one gets (180 degrees — β) the distance in degrees of the focus from the anterior pole of the eye (see table 2).

I named this instrument (see figure 3) the graduated peilingator (because its scale is divided in degrees), according to the new expression, peilingation, suggested by Tzeekoolenko. It consists of a thin metallic ring with a 14 mm. diameter of the clear space; the width of the ring is 3 mm. The instrument is placed on the eyeball concentrically to the cornea so that its interior surface clings close to the globular surface of the eyeball. On the exterior surface of this ring a small metallic rod is attached in such a way that its axis appears as a prolongation of the radius of the eyeball and forms an angle of 45 degrees with the optic axis.

In the upper part of this rod is a notch in which one may shift freely a small thin metallic arc; its direction may correspond to any meridian of the eyeball.

TABLE 2.—*Calculations in Degrees of the Position of the Morbid Focus on the Sclerotic*

Visual Angle, Degrees	Distance From the Posterior Pole of the Eye	Distance From the Anterior Pole of the Eye
5	8.1	171.9
10	16.3	163.7
15	24.4	155.6
20	32.4	147.6
25	40.4	139.6
30	48.3	131.7
35	56.1	123.9
40	63.8	116.2
45	71.3	108.6
50	78.7	101.3
55	85.9	94.1
60	92.9	87.1
65	99.7	80.3
70	106.0	74.0
75	112.3	67.7
80	118.2	61.8
85	123.7	56.3
90	128.9	51.5

Along the inner border of the ring, facing the limbus, at every 15 degrees are graduations; the axis of the inclined rod corresponds to the zero point of these graduations. A point is made with india ink at the upper end of the vertical diameter of the cornea, and one may then place the instrument on one of the coordinates of the morbid focus. The ring of the peilingator is placed on the eye concentrically to the cornea, and opposite the point, at 12 o'clock, is placed the line of graduation which corresponds to the meridian on which the morbid focus is located. With such placing the metallic arc which slides in the notch of the rod takes the direction corresponding to the meridian and will appear placed concentrically as regards the surface of the sclerotic. It will be everywhere at a distance of 2 mm. from the surface of the sclerotic.

The operator now pushes forward the anterior end of this arc in accordance with the graduations made on its marginal side, it being taken into consideration that the distance measured in degrees between

its end and the axis of the inclined rod will equal the value in degrees of the distance of the focus from the anterior pole (the second focal coordinate) reduced by 45 degrees (the distance in degrees of the axis of the inclined rod from the optic axis of the eyeball), and fixes it in this position by means of a screw.

The pointed indicator (the point of a pin) attached to the anterior end of the arc will almost come into contact with the sclerotic and
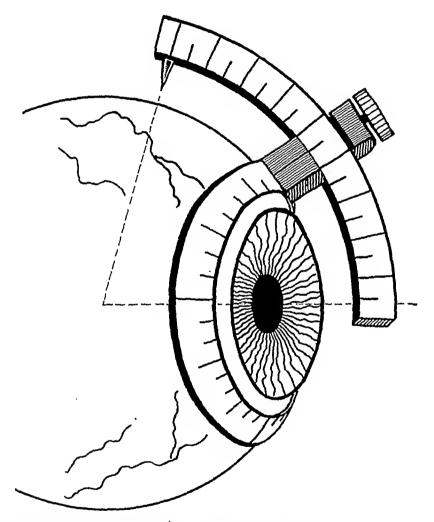
Fig. 3.—Drawing of Dashevsky's graduated peilingator. The scale is divided in degrees.

at the setting just mentioned will show the projection of the focus on the sclerotic.

The process of the peilingation of the point on the sclerotic consists of the following procedures:

1. A point is marked with india ink at the upper end of the vertical diameter of the cornea.

2. The sclerotic is laid open in the location of the focus.

3. The arc of the peilingator is fixed with a screw in a position which corresponds to the distance in degrees of the focus from the anterior pole of the eyeball.

4. The ring of the peilingator is placed on the eye concentrically to the cornea.

5. The assistant makes a mark with india ink on the sclerotic opposite the arc of the peilingator. That is the projection of the focus on the sclerotic and the exact place for the incision or the cauterization.

The apparatus is held in position on the eyeball by means of nippers which grasp the rod.

This rod will be in the region of the eyelid with any position of the eyeball. The ring of the instrument, which is not thicker than 0.85 mm., may disappear with the cornea behind the eyelids and will not hinder the rotation of the eyeball.
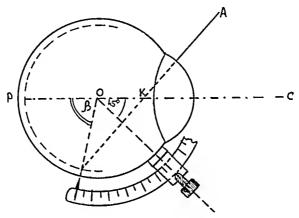
Fig. 4.—The graduated peilingator in place on reduced eye.

All the calculations for my new method are confined to the defining of the central angle, which corresponds to the visual angle, found by means of the perimeter. The first coordinate equals 180 degrees less this central angle. The second coordinate is the incline (in degrees) of the meridian on which the focus is located.

I shall now consider the helpfulness of the new method of localization and peilingation in obviating or reducing the aforementioned defect inherent in all the former methods and depending on alteration of the length of the eyeball. I shall analyze the same sample that Lindner analyzed. An eyeball with myopic refraction of 9.0 D. has an axis which is 3 mm. longer than normal. It is necessary to point out that in a person with myopia the change in the form of the eye is in the posterior segment only. Hence, if the focus is located at the equator of the eye its projection on the sclerotic will undergo no change which influences the precision of the peilingation. Two cases will now be con-

sidered. If it is supposed that the visual angle in the first case measures 30 degrees, then, according to the constants of the reduced eye, the central angle will measure 48.3 degrees. In that case the elongation of the radius will measure approximately 1.5 mm. (see figure 5). The distance from the center to the nodal point remains unchanged. The central angle B_1OP_2 is reckoned according to the sine formula

1. $\dfrac{OK}{OB_1} = \dfrac{Sin\ OB_1\ K}{Sin\ 30°}$

2. $Sin\ OB_1K = \dfrac{6.4}{11.7} \cdot Sin\ 30°.$

By aid of the mathematical table it is found that the angle OBK equals 15.9 degrees. Hence the central angle BOP equals 30 degrees + 15.9 degrees, or 45.9 degrees; i. e., because of the elongation of the axis of the eye it became diminished by 2.4 degrees (48.3 degrees — 45.9 degrees = 2.4 degrees).
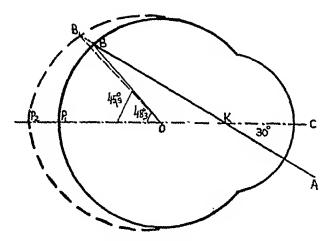
Fig. 5.—Diagram illustrating the method of calculating the central angle in a myopic eye.

Next, let it be supposed that in the second case the visual angle measures 15 degrees; i. e., the angle is located not far from the center. According to the constants of the reduced eye, the corresponding central angle will measure 24.4 degrees. In case of myopia of 9.0 D. the length of the radius will be approximately 3 mm. more and will measure 13 (10 + 3) mm.

The reckoning, made in the same way as in the former example, will show that the central angle measures 22.2 degrees; i. e., it will be diminished by 2.2 degrees. The nearer the visual angle is to the center, the smaller this difference will be, on account of the diminution of the central angle.

I am ready to consider the difference of 2.4 degrees as the highest possible one for my example. But the unit of 1 degree measures 0.25 mm. on the arc of the peilingator. Consequently, even on the arc of

the peilingator the error will not exceed 0.5 mm., but on the sclerotic this error will be much less, owing to the smaller length of the radius of its curvature. With the actual state of the surgical technic I take it for granted that the possible error will be 0.5 mm. I conclude that my method excels all others on account of these considerations and also because of the following: As was previously said, the six hundred corneas which I had measured varied in the vertical and horizontal meridians from 9.5 to 12.5 mm. Disregarding the variations of 9.5 mm. for the vertical diameter and 12.5 mm. for the horizontal diameter, which are rarely met with, I shall analyze the variations of from 10 to 12 mm. The coefficient of the correlation between the vertical and the horizontal diameter is not very great; it equals 31 per cent, but nevertheless it shows that when the one diameter is lengthened the other also has the tendency to become elongated. But what is going on in the other parts of the eyeball? One cannot answer this at present but can only make suggestions. Either the cornea becomes enlarged independently and the eyeball remains unchanged, or, in cases in which one sees a large or a small cornea, examination may show that the other parts of the visual organ have also undergone alterations as regards enlargement or diminution. The second hypothesis appears to be the more natural and probable one. It is indirectly corroborated by the results of the direct measurements. Tron made facometric observations on fifty-three emmetropic eyes and became convinced that the optic axis varies greatly. He found that 19.02 and 26.43 mm. are the extreme dimensions of the optic axis.

It is obvious that not only the length of the axis of the eyeball had been altered but that the eyeball as a whole was of greater or smaller dimensions in the various cases.

Hence it is also obvious that with such variations the length of the radius of the surface of the eyeball, on which all authors measure the distance from the limbus, also becomes altered. Because the slightest alteration of the radius results in the alteration of the length of the surface, one and the same distance in degrees will be shown by different numbers in millimeters, measured on the sclerotic.

But I must point out here that with the equal change of the size of the eyeball and of all its parts the correlation of these parts cannot be changed, and therefore the units in degrees remain unaltered.

The method that I have proposed is based merely on the measurement in degrees of the coordinates of the focus on the eyeground, and for the purpose of peilingation coordinates are taken which are shown in the angle units.

Let figure 6 be considered. The distance from the center of the graduated ring of the peilingator to the optic axis equals 7.92 mm. With

a radius of 11.2 mm. (i. e., 10.2 mm. long, according to Werbitzky + 1 mm. for the thickness of the wall of the eyeball) the angle between the optic axis and the axis of the rod of the peilingator measures exactly 45 degrees. Will it be changed if the length of the radius of the surface of the eyeball becomes changed? Let the extent of these changes be analyzed. In the first place, one must know the length of the radius that one can encounter. For a radius of 11.2 mm. I assume that the size of the corneal diameter is 10.74 mm.[2]

With the aid of simple calculations (based on the proportional changes of the corneal diameter and of the radius of the surface of the eyeball) I obtained the angles shown in table 3.

With the formula $\frac{7.92}{r} = \mathrm{Sin}\ I$ reckoned the size of the angle δ between the optic axis and the axis of the rod of the peilingator. It
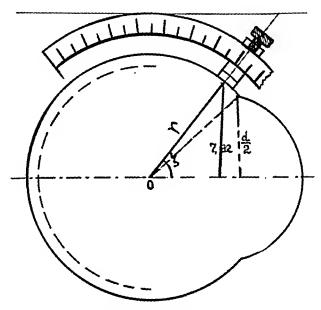
Fig. 6.—Diagram illustrating the method of calculating the inclination of the rod of the peilingator in accordance with changes in the radius of the eyeball.

appears that these changes are not great (see table 3). Considering that on the arc of the peilingator each degree equals 0.25 mm. and on the sclerotic it is much less, I am sure that my peilingator can be used with a correction when the corneal diameter varies from 10 to 12 mm. With a corneal diameter of 10 mm. it is necessary to regard the rod of the peilingator shifted to 50 degrees, with a corneal diameter of from 10.5 to 11 mm. to regard it shifted to 45 degrees and with a corneal diameter of from 11.5 to 12 mm. to regard it shifted to 40 degrees. This means

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2. In accordance with my measurements the average length of the horizontal meridian equals 11.13 mm., and that of the vertical meridian 10.36 mm.; $\frac{11.13 + 10.36}{2} = 10.74$.

that with such variations of the corneal diameter the readings on the arc of the peilingator may be changed by 5 degrees. In other respects the process of peilingation remains unchanged.

The method of the localization and peilingation by degrees appears thus to be a unique method which permits one to take into consideration the different variations of the size of the visual organ.

The first case in which the reckonings in my table of the distances from the limbus and the new method with the use of the graduated peilingator were applied and verified corroborated their exactness.

REPORT OF CASE

G., a worker, had a trauma of the left eye six months before examination. The metallic foreign body remained in the eye during this period. The eye was quiet. Vision began to be reduced and was 0.2 on examination. Ophthalmoscopic examination showed a large metallic splinter, one end of which was firmly fixed in the wall of the eyeball at the ora serrata, the place of piercing. The other end

TABLE 3.—*Angles Obtained on the Basis of Proportional Changes of the Corneal Diameter and of the Radius of the Surface of the Eyball*

Corneal diameter (d), mm.	10.00	10.50	10.74	11.00	11.50	12.00
Radius of the surface of the eyeball (r), mm.*..	10.43	10.95	11.20	11.47	11.98	12.51
Angle δ, degrees	49.40	46.30	45.00	43.70	41.40	39.30

* It is necessary to point out that when the angles obtained by me are compared as regards the extreme variations of the corneal diameter, namely, r = 9.03 for the diameter of 9.5 mm. and r = 13.04 for that of 12.5 mm. obtained by Tron, there appears to be a coincidence: The optic axis, according to Tron, varies from 19.02 to 26.43 mm., and according to my calculations 2r varies from 18.06 to 26.08 mm.

of the splinter hung loosely in the vitreous and was surrounded by flowing opacities. In the place where the splinter was attached one saw a white atrophic focus and a deposit of black pigment. Examination with the perimeter showed that the coordinates of the fixed end of the fragment were equal. 1. The visual angle (perimetrically determined) was 87 degrees. 2. The incline over the horizontal meridian of the eyeball was 10 degrees. The angle γ was positive and equaled 6 degrees. Subsequently the real visual angle (from the optic axic of the eyeball) equaled 87 degrees — 6 degrees, or 81 degrees. According to table 1, the distance from the limbus equaled 6.5 mm. By the method of Donders it equaled 9.5 mm., and by the method of Levitsky, 8 mm. It has been stated that the splinter was fixed at the ora serrata. The end of the ora serrata is generally 6 or 7 mm. from the limbus. Then were reckoned the coordinates for the graduated peilingator. The central angle (β) of 119 degrees corresponded to the visual angle of 80 degrees; hence the distance in degrees of the point sought for from the anterior pole of the eyeball was 61. When the arc of the peilingator was placed at 61 degrees it became evident that the distance of the point of the indicator from the limbus must equalize 6.5 — 7 mm. Thus, the measurements in degrees and the linear measurements, shown in my table, coincided.

On Nov. 25, 1935, an operation in which a magnet was used was performed (by Prof. D. M. Natanson). When the incision was made and the conjunctiva was separated the place where the wall of the eyeball was pierced appeared distinctly on the sclerotic as a dull and rather tender scar. The direct measurement

of its distance from the limbus was almost 7 mm. The point of the indicator of the graduated peilingator, placed on the eyeball, showed distinctly the place where the sclerotic was pierced. A small incision was made in this place, and by the aid of a hand magnet a metallic splinter 11 ·mm. thick and 7 mm. long was extracted.

Comment.—In this case ophthalmoscopic perimetry and natural control in the form of finding on the sclerotic a point sought for were successfully combined, and the advantage of the accurate reckoning based on the measurements of Werbitsky's reduced eye and of · my precise method of localization and peilingation with the use of the graduated peilingator was distinctly shown.

BIBLIOGRAPHY

Booshmich, D. G.: Sovet. vestnik oftal. **5**:185, 1934.

Dashevsky: Tr. Ukrain. Leonard Hirshman Mem. State Ophth. Inst. **3**:151, 1935.

Fischer: Zentralbl. f. d. ges. Ophth. **25**:1, 1931.

Guist: Ztschr. f. Augenh. **72**:230, 1930; cited by Lindner, K.: Wien. klin. Wchnschr. **43**:225, 1930.

Goldfeder, A. E.: Arch. oftal. **1**:547, 1926.

Graefe, A.: Arch. f. Ophth. (pt. 1) **28**:187, 1882.

Haase, G.: Klin. Monatsbl. f. Augenh. **61**:324, 1918.

Lazarev, E. G.: Sovet. vestnik oftal. **5**:161, 1934.

Lenz, in von Elschnig, A.: Augenärztliche Operationslehre, Berlin, Julius Springer, 1922, vol. 2.

Levitzky: Russk. oftal. j., 1926, nos. 8-9, p. 825.

——and Goldman: Ukrain. med. Visti, 1927, no. 1, p. 17.

Lindner: Arch. f. Ophth. **123**:233, 1929.

Loginov: Russk. oftal. j. (nos. 2-3) **12**:192, 1930.

Lossev: Russk. oftal. j., 1928, vol. 9.

Natanson, D. M.: Sovet. vestnik oftal. **5**:41, 1934.

Odintzov and Orlov: Manual of Eye Surgery, Moskva Medgia, 1934.

Reitsch: Klin. Monatsbl. f. Augenh. **87**:145, 1931.

Report concerning the Thirteenth International Ophthalmological Congress, Russk. oftal. j. **11**:261, 1930.

Rochat, G. F.: Klin. Monatsbl. f. Augenh. **91**:411 (Sept.) 1933.

Salzmann, M.: Arch. f. Ophth. **123**:252, 1929.

Tron, Eugen: Arch. f. Ophth. **126**:454, 1931; Zentralbl. f. d. ges. Ophth. **25**:782, 1931.

Tzeekoolenko: J. nautshno-issledov. kafedri (no. 5) **2**:67, 1925; Russk. oftal. j. **11**:747, 1930; **13**:302, 1932.

Vasjutinsky, A. G.: Russk. oftal. j., 1926, nos. 8-9, p. 832.

Werbitzky: Russk. oftal. j. **8**:210, 1928.

Weve: Klin. Monatsbl. f. Augenh. **87**:145, 1931.

BILATERAL METASTATIC CARCINOMA OF THE CHOROID

REPORT OF A CASE

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NEW YORK

Metastatic carcinoma of the choroid and other ocular structures is of sufficiently rare occurrence to make the following report of interest. Carcinoma involving the intra-ocular and extra-ocular tissues is considered a secondary growth arising from a primary neoplasm situated elsewhere in the body. In about 70 per cent of such cases the primary growth is carcinoma of the mammary gland, other primary foci being the lungs, the stomach, the prostate and other organs. Sometimes the primary growth cannot be located during life and is revealed only by a general autopsy.

The ocular lesion may be the only visible evidence of metastasis; hence a systematic ocular examination should be performed in all cases of carcinoma. In this condition the media are clear, permitting a distinct view of the fundi. The characteristic finding is the choroidal tumor, which is flat and grayish. It is situated temporally around the posterior pole, where it is thickest, and sometimes extends from the ora serrata to and around the disk. It is covered and surrounded by a flat retinal detachment, which obscures the visibility of its contour. The carcinomatous tissue may invade one or several ocular structures, or these foci may occur independently of each other. Differentiation between them is usually impossible, even in serial sections of the eye.

Transillumination is frequently of no avail, owing to the posterior location of the growth. Tonometric examination is also of little value, since the tension is not increased until the occurrence of late complications. When the retrobulbar tissue becomes markedly involved there will be disturbed motility and exophthalmos.

The rapid development of the choroidal tumor causes detachment of the retina, resulting in a sudden marked deterioration of vision which in one fourth of such cases is bilateral. Simultaneous involvement of both eyes is rare; first one becomes affected and then the other. When the disturbance in vision is bilateral, a tumor can generally be recognized in each eye. It is usually impossible to differentiate between

a unilateral flat carcinoma without a known primary growth and a flat sarcoma of the choroid. However, if clinically the growth suggests a flat sarcoma, enucleation in order to establish a diagnosis and prognosis should be considered.

The cancer cells are transported by the blood stream from the original focus to the ocular structures. These cells form emboli in the ocular vessels, and the emboli finally break through the walls of the vessels into the surrounding tissues.

Metastatic choroidal carcinoma occurs mainly in women, and the growth in both men and women usually appears between the ages of 30 and 60. The lapse of time between the occurrence of carcinoma of the breast and the appearance of the ocular manifestations is variable; it may be weeks or years. The outlook for life, even when the lesion is unilateral, is unfavorable; when it is bilateral the prognosis is even graver. In the former case death may be delayed for a few years; in the latter it is seldom delayed more than a few months.

Treatment is generally palliative, since the condition is a generalized one (carcinomatosis). In a very few cases radium or roentgen treatment has been tried with some success.

The typical pathologic change in the ocular structure consists principally in the presence of cancerous cellular elements. These are similar to those observed in the primary growth, but their arrangement varies according to the nature of the tissues which they invade or infiltrate. In the choroid the cellular type of carcinoma predominates: The cells replace the vessels of the choroid, arranging themselves in strands or cords, and small nests of epithelial cells form alveoli sparsely surrounded by fibrous tissue. The form of growth invading the retrobulbar region is usually the alveolar or scirrhous type of carcinoma. The sclera, optic nerve and sheath may be similarly involved, while the retina, iris and ciliary body are affected.

REPORT OF CASE

History.—F. J., a single woman aged 27 years, was admitted to the New York Skin and Cancer Unit of the Post-Graduate Medical School and Hospital on July 17, 1934, complaining of local pain caused by a mass in the left breast. The pain was first felt in September 1933. Her family history and her personal history had no bearing on the present condition. She was pale and obese. A complete physical examination revealed nothing abnormal with the exception of the condition of the breast. The right breast was normal. In the left breast a large mass could be felt in the outer half; it was hard, diffuse and somewhat infiltrated. Each axilla, but especially the right, showed enlarged nodes. A diagnosis of carcinoma of the left breast was made, and the patient was advised to enter the hospital for an operation. Radical mastectomy was performed on August 10.

The pathologic diagnosis of the removed tumor was scirrhous carcinoma of the breast (grade 2). Microscopic examination of sections of the breast showed a growth of epithelial cells arranged in nests and surrounded by a fibrous stroma (figs. 1 and 2). The nodes showed a similar structure (fig. 3).
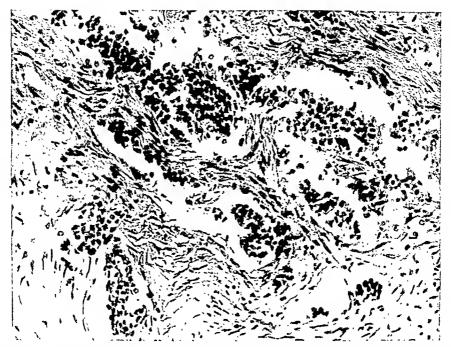
Fig. 1.—Photomicrograph (medium high power magnification) of the tumor of the breast, showing nests of tumor cells separated by edematous fibrous tissue.
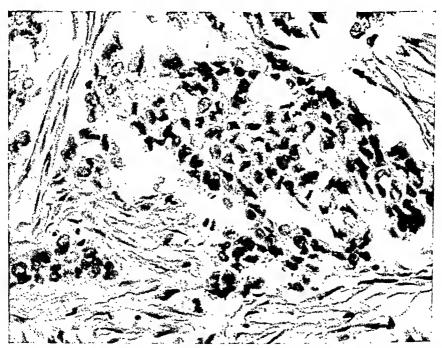
Fig. 2.—Photomicrograph (high power magnification) of an area from the tumor of the breast, showing large carcinomatous cells.

Ten days after the operation the wound became infected. The infection lasted four days, after which the wound healed. The patient remained in the hospital one month. During this time she complained of pain in the right sacro-iliac region, but roentgen examination of this area yielded negative results. After being discharged from the hospital she frequently returned for roentgen therapy. At her last visit to the clinic she complained of marked visual disturbances in each eye and was thereupon referred to the ophthalmic department of the New York Post-Graduate Medical School and Hospital.

Report of Ophthalmologic Examination.—The patient was admitted to the ophthalmic ward on Oct. 15, 1935. Four months before this date vision of each eye had suddenly become defective, first one eye being affected, then, a little later, the other. The visual disturbance was accompanied by general malaise, cough and pain in the right hip joint. Physical examination gave negative results, and the
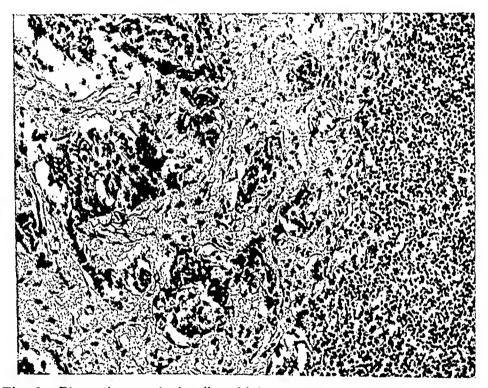
Fig. 3.—Photomicrograph (medium high power magnification) showing metastatic focus of carcinoma in a regional lymph node, surrounded by lymphatic infiltration.

laboratory reported no abnormality. After a roentgen examination and study of the lungs, Dr. H. W. Meyer diagnosed the condition as a metastatic tumor secondary to a tumor of the lungs.

The external appearance of the eye was normal with the exception of moderate dilatation of the pupils, which reacted sluggishly to light and in accommodation. Vision was eccentric and was equal in the left eye to 5/200 and in the right to 3/200. The field of vision was faulty in each eye but could not be accurately determined, owing to lack of cooperation. No exophthalmos or loss of motility could be observed in either eye. In each eye the intra-ocular tension was normal, and the ocular media were clear.

The lesion in the fundus present in each eye was significant, consisting of a tumor in the choroid. The tumors in the two eyes were practically identical in

configuration, size, color and location (fig. 4). The mass was flat, sharply cir-
cumscribed, grayish and moderately pigmented. The tumor was covered by a flat
retinal detachment, located posteriorly in the upper temporal and nasal area of the
fundus. The choroidal growth in each eye was elevated 3 mm. The optic disk
and retinal vessels appeared normal, and no hemorrhages or exudates were visible.
The diagnosis of the fundic condition was bilateral carcinoma of the choroid
(metastatic).

Course and Outcome.—During the patient's stay of one month at the hospital
the aforementioned picture of the fundi remained practically stationary. She was
discharged and transferred to the Bellevue Hospital, in the service of Dr. Webb
Weeks, on November 27. I was given the opportunity of examining her fre-
quently. After a short stay in the hospital she became cachectic, and the ocular
condition became aggravated. In the right eye, vision was reduced to that in
amaurosis. The pupil became widely dilated and fixed, the ocular conjunctiva
chemotic and the fundus ischemic, and the retinal detachment seemed to be total.
The tumor was now elevated 4 mm. The disk showed moderate papilledemas
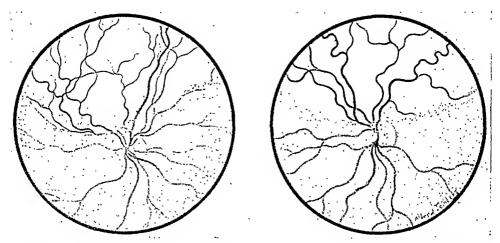
Fig. 4.—A drawing of the fundus of each eye on admission, showing flat
bilateral retinal detachment due to bilateral metastatic carcinoma of the choroid.

surrounded by numerous small hemorrhages. The retinal veins became engorged
and tortuous; the arteries became narrowed, and the intra-ocular tension rose
to 56 mm. of mercury (Schiötz). The exophthalmometric reading was 20 mm.,
and the motility of the eye was restricted in all directions, causing pain on motion.
The cornea, anterior chamber and media were clear. In the left eye, vision was
reduced to perception of light, and there was faulty projection. The lids, cornea,
anterior chamber and iris were normal. The pupil was widely dilated and fixed.
Examination of the fundus revealed two large retinal detachments in the lower
quadrant and a recent one in the upper temporal area. The circumscribed mass
visible above the disk was elevated 5 mm. and retained its grayish color, but its
outline was less distinct. No hemorrhages or exudates were present. The disk
was hyperemic and elevated, indicative of incipient papilledema. The media
remained clear and the retinal vessels normal. The intra-ocular tension was
29 mm. of mercury (Schiötz), and the exophthalmic reading was 18 mm. The
diagnosis of the fundic condition was bilateral carcinoma of the choroid (meta-
static), with bilateral retinal detachment and moderate bilateral papilledema.

During the patient's four month stay at the hospital several roentgen examinations were made, which showed metastatic foci in the lungs, skull, ribs and long bones. She gradually became extremely cachectic, and died on March 30, 1936. The diagnosis was carcinomatosis with metastases to the eyes.

Permission for a general autopsy was refused, but removal of the eyes was permitted. These were fixed and stained in the usual manner and were then sent to me through the agency of Dr. Edward Gresser.

Report of the Enucleated Eyes.—Right Eye: The eyeball was misshapen owing to manipulation following enucleation. Low power magnification (fig. 5) showed the anterior segment of the eye to be normal. The retina was detached. The posterior segment showed thickening of the choroid, principally on its temporal side and most prominent around the posterior pole. The choroidal growth appeared
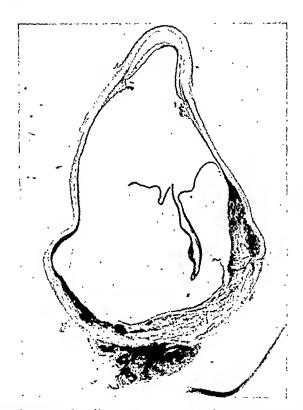
Fig. 5.—Photomicrograph (low power magnification) of the right eyeball, showing carcinomatous involvement of the choroid, sclera and retrobulbar tissue.

flattened and stretched from the disk to the region of the ora serrata. On the nasal side of the disk, extending anteriorly for a short distance, appeared a flat thickening of the choroid. The sclera showed a few dark spaces, probably cancerous foci. Surrounding the head of the optic nerve, at the apex of the retro-orbital region, were dark cancerous areas, which were more apparent on the temporal side in the neighborhood of the posterior short ciliary vessels. The head of the optic nerve, behind the lamina cribrosa, showed carcinomatous foci.

Microscopic study of sections of the eye revealed no lesions in the cornea, anterior chamber, iris or vitreous. The retina was completely detached, but its structural layers, including the lamina vitrea were normal. The important pathologic conditions appeared in the choroid (figs. 6 and 7). The vascular layers were replaced by carcinomatous tissues, arranged in acini which were filled with cancer cells. A few of their central areas showed degeneration, but no
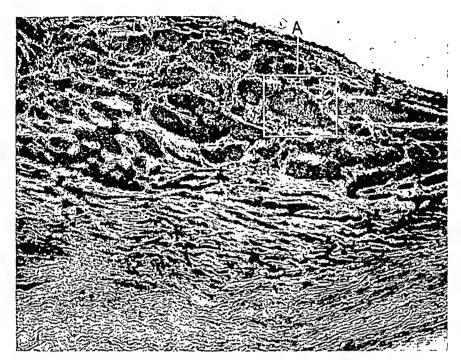
Fig. 6.—Photomicrograph (low power magnification) of the tumor in the choroid of the right eye, which resembles the tumor of the breast.
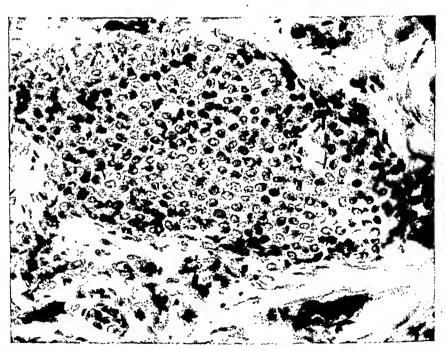
Fig. 7.—Photomicrograph (high power magnification) of area *A* of figure 6, showing slight degeneration in a cellular nest.

hemorrhages were observed. The arrangement of the cancerous tissue was mainly of the glandular type, owing to the structure of the choroid. There was some invasion of the lamina suprachoroidea. A few nests of cancerous cells were seen between the lamellae of the sclera. The stem of the fibers of the optic nerve just behind the lamina cribrosa showed a few nests of carcinoma cells, possibly transported by the central retinal artery. Surrounding the head of the optic nerve in the retro-orbital tissue (fig. 8) the cancerous invasion was marked, appearing in the vicinity of the posterior ciliary vessels. The arrangement of the cancerous tissue in this area was mainly of the alveolar type, owing to the nature and structure of the retrobulbar tissue invaded by the metastatic process. Degeneration of the cells was noted in the center of a few acini, but no hemorrhages were visible.
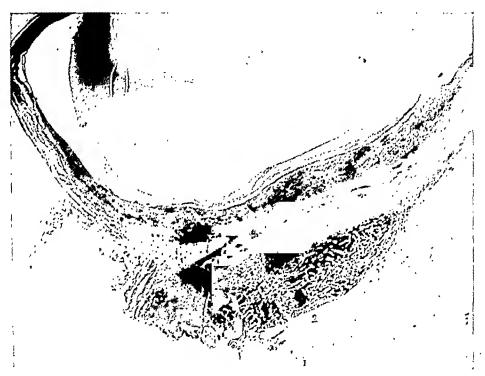
Fig. 8.—Photomicrograph (medium high power magnification) showing metastatic focus in the retrobulbar tissue and the optic nerve (right eye).

Left Eye: The eyeball (fig. 9) also was somewhat misshapen from manipulation. The media seemed clear. There was an artificial detachment of the retina. A thin mass could be seen encircling the posterior segment of the eye, and there was a thicker mass posteriorly in the retrobulbar region.

Microscopic examination showed nothing abnormal in the cornea, anterior chamber, iris and lens. The layers of the detached retina, including the pigment epithelium and the lamina vitrea, also appeared normal. The choroid was replaced by carcinomatous tissue, thinned out and extending from the ora serrata on the nasal side to the ora serrata on the temporal side. The flat growth was extremely cellular and apparently glandular in type. Some of the vessels in Sattler's layer were still recognizable. The choriocapillaris was completely replaced by carcinomatous tissue, and vessels contained carcinomatous cells (fig. 10). A few chromatophores were visible. There was some invasion of the lamina suprachoroidea, and the scleral spaces contained many nests of carcinomatous tissue. The retro-
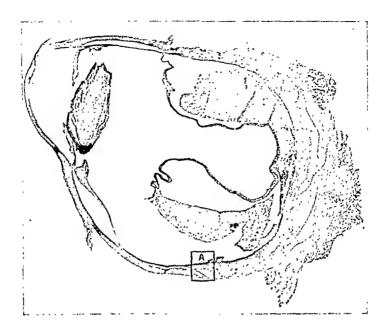
Fig. 9.—Photomicrograph (low power magnification) of the left eyeball, showing carcinomatous involvement of the choroid, sclera and retrobulbar tissue.
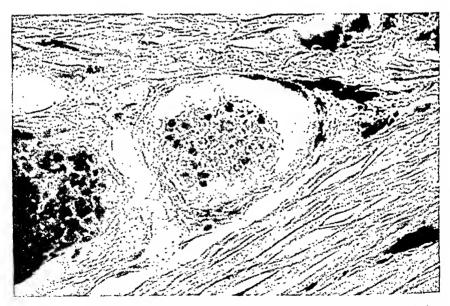
Fig. 10.—Photomicrograph (high power magnification) of area *A* of figure 9, showing an enlarged blood vessel containing carcinomatous cells.

orbital tissue surrounding the optic nerve head was markedly infiltrated with carcinomatous tissue of the alveolar type, and some of the smaller capillaries of this region were filled with carcinoma cells.

Pathologic Diagnosis.—This diagnosis was bilateral carcinoma of the choroid, with involvement of other ocular structures (metastatic).

COMMENT

Bilateral metastatic carcinoma of the choroid is rare; unilateral involvement occurs more often. In more than one fourth of the cases of the latter type the condition ended in bilateral ocular metastases.

Carcinomatous metastases may occur in one or several ocular structures, owing to an extension of the primary metastasis or to independent foci.

The visibility of the choroidal growth is often obscured by a flat retinal detachment. The finding of a primary carcinoma elsewhere in the body would suggest that the retinal detachment was caused by metastatic choroidal carcinoma.

An ophthalmologic examination is suggested in cases of primary carcinoma occurring in any part of the body, as the ocular lesion may be the only evidence of metastasis.

Further reports of ocular metastases are necessary in order to establish by statistics the relationship between these metastases and any primary carcinoma.

OCULAR DISTURBANCES ASSOCIATED WITH EXPERIMENTAL LESIONS OF THE MESENCEPHALIC CENTRAL GRAY MATTER

WITH SPECIAL REFERENCE TO VERTICAL OCULAR MOVEMENTS

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The region around the aqueduct of Sylvius is of special interest from an ophthalmologic point of view. One has to deal here not only with the nuclei of the third and fourth cranial nerves, below the anterior and the posterior quadrigeminal body, respectively, and with their afferent fibers for the optic and the vestibular reflex, but also with descending pathways, which transmit impulses from the hypothalamic centers of the vegetative system to the cervical portion of the sympathetic system and finally the tectal region and the nuclei of the central gray matter, the function of which is still rather obscure. Clinically it is known that lesions of the region induce anomalies of the pupillary innervation, disturbances of accommodation, and convergence, conjugate deviation of the eyeballs and paralysis of associated movements, particularly in the vertical direction. The pathologic processes producing these symptoms are, unfortunately, usually not sharply limited; they are tumors acting not only by localized destruction but also by pressure, the effect of which may reach as far as the base of the brain, or inflammatory processes extending to neighboring areas. Thus questions of finer localization may not be solved by study of clinicopathologic material alone; the pathologic changes observed in man raise questions rather than give an answer to these problems. It seemed, therefore, of interest to study the ocular symptoms in experimental stimulation and destruction of the region in the vicinity of the aqueduct of Sylvius.

METHOD

The experiments were performed on thirty cats. As a rule, Clarke's stereotaxic apparatus was used for the stimulation experiments as well as for the production of circumscribed lesions. Since the brain was punctured from the dorsal aspect, in or close to the midline, in the region of the sagittal sinus, it proved advantageous to produce coagulation of this sinus by thermocautery after the skull was trephined above the sinus, so that the subsequent puncture was followed by a minimal hemorrhage only. In order to reach the central gray matter below the anterior quadrigeminal body the punctures were made in levels from 4 to 8 mm. cranial to the interaural line and reached from 19 to 23 mm. below their entrance into the surface of the hemisphere. An insulated silver wire with a bare tip was thrust into the brain, either in the midsagittal line or from 1 to 2 mm. lateral to the midline. The tip of the wire acted as the stimulating electrode, and a plate applied to the abdomen as the indifferent electrode. For the following electrolytic destruction the wire was connected with the anode of a source of direct current, and a current of from 1 to 3 milliamperes was applied for from two to three minutes. In a number of experiments lesions were produced by injection of small amounts (from 0.05 to 0.2 cc.) of a 70 per cent solution of alcohol or of a 20 to 40 per cent solution of formaldehyde instead of by electrolytic destruction. For this purpose a thin injection needle with a square end was placed in the stereotaxic apparatus instead of the silver wire. The injected fluid was stained with methylene blue (methylthionine chloride) or with eosin. After study of pupillary reactions and ocular movements for one or several days following production of the lesion, the frontal and the occipital center for ocular movements on each hemisphere were exposed and stimulated. In order to facilitate observation of the ocular reactions an incision was made into the external canthus, the nictitating membrane was excised, and the eyelids were widely separated. The extent of the lesions of the midbrain was studied on serial sections stained with Weil's stain or by the Loyez method.

COMMENT

Stimulation of the gray matter may yield constriction as well as dilatation of the pupil, the former symptom particularly if the electrode lies in the level of the posterior commissure and the latter symptom also if the more posterior parts are stimulated. Yet these observations should not be interpreted as indicating that the reflex arc of the pupillary light reflex takes its way over the mesencephalic gray matter, as some clinicians (Wilson[1] and Redlich[2]) supposed.

Spiegel and Nagasaka[3] showed some years ago (1926) that destruction of the gray matter around the aqueduct of Sylvius does not abolish the light reflex of the pupil. Ranson and Magoun's[4] stimulation experiments also indicate, as shown by their schematic diagram, that the reflex

1. Wilson, K.: J. Neurol. & Psychopath. **2:**1, 1921.
2. Redlich, E.: Wien. klin. Wchnschr. **35:**756, 1922.
3. Spiegel, E., and Nagasaka, G.: Arch. f. d. ges. Physiol. **215:**120, 1926.
4. Ranson, S. W., and Magoun, H. W.: Central Path of Pupilloconstrictor Reflex in Response to Light, Arch. Neurol. & Psychiat. **30:**1193 (Dec.) 1933.

arc of the pupillary light reflex takes its way outside the central gray matter, the pathway of the pupilloconstrictor fibers to the nucleus innervating the sphincter muscle arching around the central gray matter.

Thus the constriction of the pupil observed on stimulation of the outer parts of the central gray matter in the level of the posterior commissure is not a local symptom of stimulation of the gray matter but is due to stimulation of the surrounding pupillary reflex arc.

The pupillodilator effects observed on stimulation of the central gray matter are explained by the fact that the pupillodilator fibers originating in the hypothalamus descend in the ventral and the lateral aspect of the central gray matter around the aqueduct of Sylvius (Beattie, Brow and Long[5]). It should be emphasized that these stimulation effects are observed, as a rule, in each eye, even if the stimulating electrode has a paramedian position and a threshold stimulus is applied. This is in agreement with the view that the pupillodilator tracts (Karplus and Kreidl[5a]), as well as the constrictor pathway (Ranson and Magoun[4]), partially decussate peripheral to the point of stimulation.

After lesions of the mesencephalic central gray matter the pupillary light reflex may recover rather quickly, often on the first or second day after operation, even if the lesion is located in the region of transition between the mesencephalon and the diencephalon, the so-called pretectal area. Quantitative changes of the light reflex may, however, be observed, if the lesion reaches the vicinity of the sphincter nucleus or the afferent part of the optic reflex arc. Spiegel and Nagasaka[3] observed in cases of unilateral lesions of the anterior quadrigeminal bodies extending into the central gray matter a transient sluggish pupillary reaction on the side of the injury. Similar and more pronounced transitory changes of the pupillary reaction were observed in the present series of experiments. Not only was the reaction to light sluggish, but the contraction of the sphincter muscle continued for several minutes after the illumination of the eye had been discontinued. In other words, the reaction had a definitely tonic character. In this stage the pupils sometimes, under faint, diffuse illumination, were rather small (from 1 to 2 mm. in diameter). These findings may be interpreted in agreement with those of Magoun and Ranson,[6] who reported similar observations as due to a transitory stimulating effect of the lesion on the reflex arc in question, particularly since these phenomena

5. Beattie, J.; Brow, G. R., and Long, C. N. H.: Proc. Roy. Soc., London, s.B **106**:253, 1930.

5a. Karplus, I. P., and Kreidl, A.: Arch. f. d. ges. Physiol. **143**:109, 1911.

6. Magoun, H. W., and Ranson, S. W.: Central Path of Light Reflex: Study of Effect of Lesions, Arch. Ophth. **13**:791 (May) 1935.

were in our cases still more short lived (as a rule, from one to two days' duration) than in those of these authors, who observed a duration up to two weeks. We may add that psychic excitation produced by painful stimuli or electric stimulation of the frontal lobe could counteract this tonic contraction of the sphincter muscle but that its relaxation was somewhat less prompt and less pronounced than normally.

It seems unnecessary to emphasize that the permanent miosis observed in the Argyll Robertson pupil may not be explained in a similar way as these transitory phenomena. We refer in this regard to · our previous discussion [7] of the Argyll Robertson pupil.

As to the innervation of conjugate ocular movements, it was shown in former experiments that after severance of the posterior longitudinal fasciculus (Spiegel and Tokay [8]) or after total transverse section of the brain stem behind the midbrain (Spiegel and Scala [9]) cortical stimulation no longer induces horizontal conjugate movements but induces only vertical conjugate movement or convergence movements of the eyeballs. These experiments showed that the essential mechanisms for the innervation of vertical ocular movements should not be sought in parts of the rhombencephalon, as supposed by some (for instance, the vestibular nuclei [Ohm [10]] or in the nucleus tecti of the cerebellum and the inferior olive [Muskens [11]]). While the cortical impulses innervating horizontal conjugate deviation have to take their way over the rhombencephalon, the connections of the forebrain with the mesencephalon proved to be sufficient for the cortical innervation of vertical ocular movements.

Continuing these experiments, we tried to determine the more exact position of the mechanisms that transmit the cortical impulses to the nuclear centers for the elevator and the depressor muscles of the eyeball. Clinically it is well known that paralysis of conjugate vertical ocular movements, so-called Parinaud's syndrome, occurs in diseases affecting the corpora quadrigemina or neighboring areas (the pineal gland, the

7. Scala, N. P., and Spiegel, E. A.: Pupillary Reactions in Combined Lesions of Posterior Commissure and of Pupillodilator Tracts, Arch. Ophth. **15**:195 (Feb.) 1936.

8. Spiegel, E. A., and Tokay, L.: Arb. a. d. neurol. Inst. a. d. Wien. Univ. **32**:138, 1930.

9. Spiegel, E., and Scala, N. P.: Cortical Innervation of Ocular Movements, Arch. Ophth. **16**:967 (Dec.) 1936.

10. Ohm, J.: Ztschr. f. Hals.-Nasen-u. Ohrenh. **39**:136, 1935.

11. Muskens, L. J. J.: (*a*) J. Comp. Neurol. **50**:289, 1930; (*b*) Deutsche Ztschr. f. Nervenh. **115**:81, 1930; (*c*) Das supra-vestibuläre System bei den Tieren und beim Menschen, Amsterdam, N. V. Noord-Hollandsche Uitgevers Maatschappij, 1934.

posterior part of the corpus callosum or of the thalamus and the cranial part of the pons). There exists, however, considerable difference of opinion regarding the interpretation of this symptom. Some have contended that symmetrical lesions in parts of the nuclei for the ocular muscles or of associational fibers may induce isolated paralysis of vertical ocular movements (Wilbrand and Saenger [12]) and that the assumption of the existence of supranuclear centers for these movements is not justified.

One may suppose, according to Spiller,[13] that certain fibers uniting parts of the oculomotor nuclei with one another or uniting the center for the inferior rectus muscle with that for the fourth nerve have a coordinating function similar to that of the posterior longitudinal fasciculus and that this function may be disturbed by small lesions. This view admits the existence of a coordinating system but leaves it undetermined where the fibers uniting the various parts of the nuclei for the ocular muscles take their origin. Holmes [14] considered it improbable that a nuclear lesion should destroy the cells innervating the elevator and depressor muscles on each side without affecting the other muscles. According to his view, this holds true particularly for the depressor muscles (the inferior rectus and the superior oblique muscles), which are innervated from two distinct nuclei separated by a relatively considerable space. Thus he arrived at the conclusion that supranuclear associational or coordinating mechanisms for up and downward movement must exist.

These supranuclear centers are generally supposed to be located in the neighborhood of the nuclei for the ocular muscles (Bielschowsky [15])—in the tectum, in the central gray matter around the aqueduct of Sylvius or in the tegmentum. Holmes inclined to the opinion that they lie in the anterior quadrigeminal bodies but arrived at no definite conclusion. Similarly, Riley [16] held that the superior colliculus controls vertical ocular movements, either through a center or through fiber pathways that pass through the colliculi. On the basis of studies in comparative anatomy, Muskens [11c] assumed that nuclei located in the gray matter around the aqueduct of Sylvius regulate the innervation of

12. Wilbrand, H., and Saenger, A.: Neurologie des Auges, Munich, J. F. Bergmann, 1921, vol. 8, p. 93.

13. Spiller, W. G.: J. Nerv. & Ment. Dis. **32**:417, 1905.

14. Holmes, G.: Brit. J. Ophth. **5**:241, 1921.

15. Bielschowsky, A.: Störungen im Augenbewegungsapparat, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1936, vol. 4, p. 173.

16. Riley, H. A.: Central Nervous System Control of Ocular Movements and Disturbances of This Mechanism, Arch. Ophth. **4**:640 (Nov.); 885 (Dec.) 1930.

vertical ocular movements. He distinguished a nucleus lateralis transmitting the impulses for downward movements and a nucleus medialis innervating the upward movements, but without proving that these nuclei have more importance for the mechanism of vertical movements than the centers postulated by him in the inferior olive and in the cerebellum. On the basis of the results of stimulation experiments Godlowsky [17] recently located the apparatus for upward movements and that for downward movements in two different regions, in the area of the posterior commissure and in the ventral part of the tegmentum, respectively. The nucleus of the posterior commissure he regarded as the supranuclear center for downward movements and the nucleus reticularis ventralis tegmenti as that for upward movements. Unfortunately, he studied only the effect of stimulation and the fiber degeneration of various tracts, without testing his theory by ascertaining the effect of subcortical lesions on the cortical innervation of ocular movements.

In our experiments stimulation of the central gray matter yielded downward movements of the eyeballs if the electrode was placed in the pretectal region or behind this level. The reaction appeared in each eye also if the electrodes lay lateral to the midline, indicating that the system that was stimulated had an effect on the homolateral and on the crossed nuclei for the ocular muscles. In about one half of the cases the downward movement was associated with dilatation, in the other half with constriction, of the pupils. Thus the supranuclear mechanism for these movements must be in the vicinity of the descending vegetative hypothalamic tracts as well as in that of the pupillary light reflex arc. From the point of view of finer localization, the combination of downward movement and convergence is of interest. It could be shown that the system stimulation of which with threshold currents induces downward movements lies dorsal to a point stimulation of which yields convergence movements. In cat 9 (fig. 1), for instance, stimulation at a distance of 21 mm. from the surface elicited downward movement of the eyes and dilatation of the pupils (the distance of the coils of the Harvard inductorium with two dry cells was 10 cm.); at a distance of 22 mm. we obtained horizontal inward movement of the right eye and inward rotation of the left eye, and at a distance of 23 mm. from the surface inward movement of both eyes. Several times the downward movement of the eyes was associated with movement of the head in the same direction, and in one case (cat 12) also with closing of the mouth. The first-mentioned combination seems of

17. Godlowski, W.: Les centers sous-corticaux du regard: Etude expérimentale, Krakow, 1936.

interest in view of the not infrequent clinical observations in epidemic encephalitis of a combination of oculogyric crises accompanied with tonic movements of the head in the same direction. One may suppose that the systems descending from the central gray matter or its neighborhood in the level of the posterior commissure not only unite the parts of the nuclei for the ocular muscles innervating vertical movements with one another but also reach the segmental nuclei innervating movements of the head in the same plane.

More rarely than downward movements, upward deviation was observed. This reaction appeared often combined with conjugate devia-
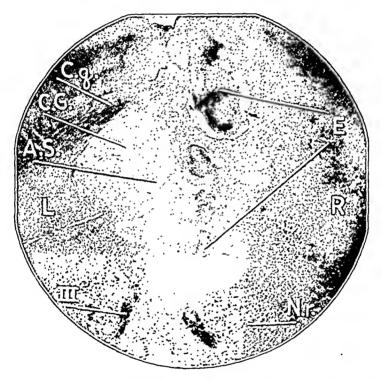
Fig. 1 (cat 9).—*E* indicates the puncture canal through the right corpus quadrigeminum (*C. q.*) and the right half of the central gray matter (*C. G.*), surrounded by the electrolytic lesions; *A. S.,* the aquaeductus Sylvii; *III,* the oculomotor nerve; *N. r.,* the nucleus ruber; *L,* the left side,, and *R,* the right side.

tion to the opposite side when the electrode was not moved far enough downward to reach the level of the aqueduct of Sylvius and medial parts of the tectum were stimulated and, particularly, when the electrode lay more anteriorly, reaching caudal parts of the optic thalamus. This is in agreement with the statment of Godlowski [17] that stimulation of medial parts of the anterior quadrigeminal body and of internal parts of the optic thalamus yields upward deviation of the eyeball. In this connection clinical observations of Holmes [14] are of interest; he reported that tumors or lesions extending from before backward into the midbrain affected first upward, then downward, movements and eventually

convergence, indicating that the center for elevation of the eyeball lies anteriorly and that for depression posteriorly.

The appearance of vertical conjugate movements on stimulation of the cortical frontal and occipital centers was prevented by producing lesions of the central gray matter in the most cranial part of the midbrain and in the region of transition to the optic thalamus. This may be illustrated by the results of the following experiments.

EXPERIMENTS WITH LESIONS OF CENTRAL GRAY MATTER IN REGION OF AQUEDUCT OF SYLVIUS

CAT 8.—On Nov. 30, 1936, at 12 o'clock, puncture with the stereotaxic apparatus was made 4 mm. anterior to the interaural line, 1 mm. to the right of the midsagittal line and 20 mm. below the surface. On stimulation (the distance of the

PROTOCOL 1.—*Cat 8*

Time	Stimulated Area	Ocular Reactions	Other Reactions
3:25	Left frontal lobe........ (distance of coils, 7 cm.)	Each eye deviated to right	Cloni of right hindleg
3:30	Left frontal lobe........	Each eye deviated to right	
3:50	Left frontal lobe........	Each eye deviated to right	Cloni of right half of body
3:54	Right frontal lobe.......	Horizontal undulation; dilatation of pupils	
3:58	Left frontal lobe........	Left eye showed clonic jerks to right after stimulation ceased: right eye showed no definite movement	Cloni on right side
4:05	Right frontal lobe....... (distance of coils, 6 cm.)	Left eye showed clonic movements to left: right eye showed no definite movement	
4:08	Left occipital lobe.......	Each eye deviated to right	
4:10	Right frontal lobe.......	Each eye deviated to left	General cloni
4:13	Left frontal lobe........	Each eye deviated to right	General cloni
4:15	Left occipital lobe.......	Each eye deviated to right	
4:18	Right occipital lobe.....	Each eye deviated to left	

coils being 10 cm.) there were movement of the eyeballs downward and contraction of the pupils. Electrolysis (with a current of 1 milliampere applied for three minutes) was carried out in this position, in the midline and 1 mm. to the left of the midsagittal line (the other indexes were the same). After electrolysis on the left side the electrode was inadvertently lowered from 2 to 3 mm.

At 3 p. m. there was a light reflex on each side. The right pupil contracted from 7.5 to 6 mm., and the left pupil from 7 to 6 mm.

At 3:25 p. m. stimulation of the cerebral cortex was carried out (protocol 1).

Histologic Examination.—The electrodes had penetrated the posterior commissure and produced lesions in the central gray matter below the commissure on each side (fig. 2). The oculomotor nuclei and the Edinger-Westphal nuclei were intact. On more caudal sections the left electrolytic lesion was continued by a fine canal that passed through the most lateral part of the central gray matter and reached the roots of the left oculomotor nerve. The majority of the fibers of this root were, however, intact. The right oculomotor nerve was normal.

CAT 14.—On Jan. 16, 1937, puncture was made 7 mm. anterior to the inter-aural line in the midsagittal line, extending to a depth of 21 mm. Stimulation (the distance of the coils being 8, 8.5 and 9 cm.) elicited upward movement of the eyes and dilatation of the pupils. Electrolysis (with a current of 1 milliampere applied for three minutes) was carried out in the midline and 2 mm. outside the midline on each side.

On January 19 the right pupil was wider than the left, and there were a distinct reaction of the left pupil to light and an uncertain reaction of the right pupil to light.

Stimulation of the left frontal lobe (the distance of the coils being 6 cm.) elicited repeatedly conjugate deviation of the eyes to the right. In some experiments
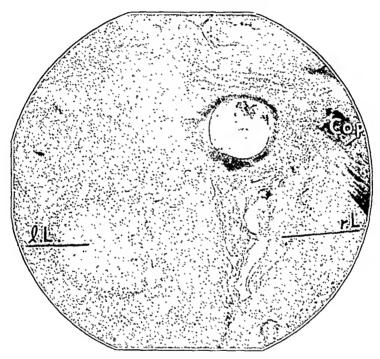
Fig. 2 (cat 8).—Electrolytic lesion in the left (l. L.) and the right (r. L.) half of the central gray matter below the posterior commissure (Co. p).

the head also turned to the right; in others there was downward movement of the head but no vertical ocular movement. Stimulation of the left occipital lobe elicited slight convergence movements.

Histologic Examination.—The maximal extent of the lesion was observed in sections through the caudal end of the third ventricle (fig. 3). Here the peri-ventricular gray matter was destroyed between the ganglia habenulae and the corpora mammillaria. Forel's field H and the cerebral peduncles were intact; on the left side the lesion extended somewhat into the medial part of the optic thalamus and the left ganglion habenulae. The lesion was seen to diminish rapidly in more caudal sections, and was limited, on section through the posterior com-missure, to the central gray matter around the aqueduct of Sylvius. The oculo-motor nuclei and nerves were not involved.

In our cases in which vertical conjugate deviation failed to appear on cortical stimulation after lesions of the central gray matter, the oculomotor nuclei were intact or, at most, injured on one side. This could be proved by the histologic examination and functionally by the fact that the tone of the sphincter muscle and in some cases also the pupillary light reflex were preserved and conjugate horizontal deviation of the eyeballs could be elicited on cortical stimulation in normal extent. The nucleus of the fourth cranial nerve was outside
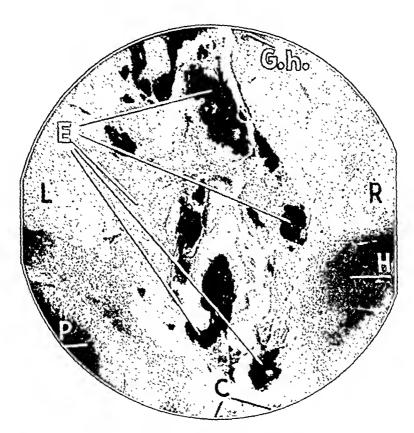
Fig. 3 (cat 14).—*E*, electrolytic lesion and hemorrhages in the central gray matter around the third ventricle, reaching from the ganglion habenulae (*G. h.*) close to the corpus mammillare (*C*). *H* indicates Forel's field H; *P*, the cerebral peduncle; *L*, the left side, and *R*, the right side.

the level of the lesions. It is, of course, possible that cells of the reticulate substance of the mesencephalic tegmentum are intercalated in parts of the pathways of the corticofugal impulses for conjugate vertical movements before these impulses reach the region of the central gray matter. The direct connection between the reticulate substance of the tegmentum and the nuclei for the ocular muscles seems, however, to be not sufficient for the cortical innervation of conjugate vertical movements in either direction, and the impulses for upward

as well as for downward movement seem to take their way over the central gray matter or closely adjacent systems before reaching the nuclei for the ocular muscles (in regard to the innervation of horizontal ocular movements see Spiegel [18]).

Small lesions of the central gray matter may induce partial loss or disturbance of vertical movements. In the following case (cat 30), for example, cortical stimulation failed to elicit upward movement of the eyeballs, although vertical deviation in either direction could be elicited by labyrinthine stimulation.

CAT 30.—On May 20, 1937, at 11 a. m. to 12, electrolysis was carried out 4 and 6 mm. anterior to the interaural line, in the midsagittal line and 1.5 mm. outside the midline on each side, the depth of puncture being 19 mm. After the lesion the eyeballs were deviated to the right, and the pupils were contracted (the transverse diameter was 2.5 mm.). There was no reaction to light (the animal was in sleep due to the injection of dial).

At 3 p. m. the animal was still in sleep induced by dial. Stimulation of the labyrinth by rotation on the Bárány chair (the head was held fixed so that the left ear was down and the right ear up) induced the following movements: After ten revolutions to the right the eyeballs deviated toward the lower lid and returned in jerky movements to the original position, and after ten revolutions to the left the eyeballs deviated toward the upper lid, then moved in opposite directions.

After exposure of the cerebral cortex on each side the eyeballs showed conjugate deviation to the left.

After stimulation of the left frontal lobe one repeatedly observed deviation of the eyeballs to the right. In one experiment this movement was combined with deviation of the head to the right; in one instance it was combined with downward movement of the eyeballs, and in another instance with convergent movement.

Stimulation of the right frontal lobe induced deviation of the eyeballs to the right or horizontal undulation combined with, or preceded by, downward movement.

Stimulation of the left occipital lobe induced only horizontal movements of the eyeballs to the opposite side.

Stimulation of the right occipital lobe induced slight horizontal undulation of the eyeballs.

Histologic Examination.—The electrode had penetrated the medial part of each anterior quadrigeminal body and produced a lesion in the dorsal part of the central gray matter (fig. 4), leaving the oculomotor nuclei and nerves intact. In the pretectal region the lesion encroached on the posterior commissure.

There was loss of the downward movement of the eyes in cat 13; the lesion was closely cranialward from the right arm of the posterior commissure and encroached on its anterior fibers. In cat 12 there was only minimal downward movement of the left eye, and distinct downward movement of the head was preserved, while upward movements of

18. Spiegel, E.: Rôle of Vestibular Nuclei in Cortical Innervation of Eye Muscles, Arch. Neurol. & Psychiat. **29**:1084 (May) 1933.

the eyes were repeatedly obtained.   The lesion was a puncture in the region of the left ganglion habenulae.   In cat 17 only impulses originating in the occipital lobe failed to produce vertical movements, while stimulation of the frontal lobe still elicited vertical movements in both directions, mostly downward but occasionally upward.   The lesion was a puncture through the medial part of the right anterior quadrigeminal body, passing alongside the right portion of the circumference of the aqueduct and encroaching on the most dorsal part of the reticulate substance outside the right posterior longitudinal fasciculus.   In cat 7 the downward movements were preserved, but instead of the conjugate
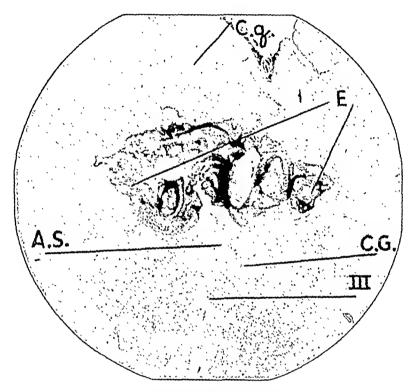
Fig. 4 (cat 30).—Electrolytic lesion (*E*) in the dorsal part of the central gray matter (*C. G.*) around the aquaeductus Sylvii (*A. S.*).   C. q. indicates the corpus quadrigeminum, and *III*, the oculomotor nucleus.

upward movements stimulation of either frontal lobe produced after the lesion in the mesencephalic gray matter skew deviation (upward movement of the right eye and downward movement of the left eye).   The lesions were bilateral punctures of the central gray matter behind the posterior commissure, more extensive on the right and encroaching on the most dorsal part of the tegmentum.   Since we observed occasionally skew deviation in former experiments [19] on stimulation of the posterior

19. Scala, N. P., and Spiegel, E. A.: Effect of Stimulation of Posterior Longitudinal Fasciculus on Ocular Muscles, Arch. Ophth. 9:939(June) 1933.

longitudinal fasciculus in the pons, it seems that this symptom may appear in diseases of various parts of the pathways innervating ocular movements and has, therefore, only limited value for local diagnosis.

In view of the fact that circumscribed lesions of the central gray matter in the cranial part of the mesencephalon may abolish the conjugate vertical ocular movements on cortical stimulation, the problem presented itself whether the pathways for this innervation reach the central gray matter by way of the tectum, which was considered by numerous authors since Adamük [20] as a coordinating center for associated ocular movements. Bernheimer [21] and Bechterew.[22] made contradictory statements. In other words, the question arose whether paralysis of vertical ocular movements in diseases of the dorsal part of the midbrain should be considered as a local symptom of a lesion of the central gray matter and its adjacent nuclei, including the tectum or excluding this ganglion.

Thus, it was necessary to ascertain in further experiments which ocular movements can still be observed on cortical stimulation of the frontal as well as of the occipital lobes after destruction of the tectum of the anterior quadrigeminal bodies. Since extensive lesions had to be produced, the stereotaxic instrument seemed less fit for these experiments. After exposure of the occipital lobes they were, therefore, gradually pushed outward by introducing small pledgets of cotton between them and the falx cerebelli and the tentorium, respectively. The sagittal sinus was ligated and cut between the ligatures, and the posterior part of the falx cerebelli was excised so that the tentorium was exposed. The vena magna Galeni and the sinus rectus were coagulated; the dura covering the tentorium was removed, and the anterior part of the tentorium was excised so that the anterior lobe of the cerebellum appeared. Then the posterior quadrigeminal bodies were gradually separated from the anterior lobe of the cerebellum so that the entrance to the aqueduct became visible. A small bent knife was thrust into the midbrain below the posterior quadrigeminal bodies, parallel to the aqueduct, separating the tectum of the posterior, and then that of the anterior, quadrigeminal bodies from the central gray matter. In some experiments the tectum of the corpora quadrigemina was destroyed by electrocautery. These operations were followed by the usual stimulation experiments on the cerebral cortex, of which the following is an example.

20. Adamük, E.: Centralbl. f. d. med. Wissensch. **8**:65, 1870.

21. Bernheimer, S.: Wien. klin. Wchnschr. **12**:1310, 1899.

22. Bechterew, W.: Funktionen der Nervenzentren, Jena, Gustav Fischer, 1909, vol. 2, p. 1041.

EXPERIMENTS WITH DESTRUCTION OF ·THE TECTUM OF THE CORPORA QUADRIGEMINA

CAT 23.—On April 14 destruction of the tectum of the anterior corpora quadrigemina by electrocautery was carried out. After this operation the right eye deviated downward, and the left eye deviated inward. The transverse diameter of the left pupil was 2.5 mm., and that of the right pupil was 3 mm.

The results of stimulation of the cerebral cortex are shown in protocol 2.

Histologic Examination.—The destruction of the tectum of the anterior quadrigeminal bodies by the electrocautery reached to the posterior commissure

PROTOCOL 2.—*Cat 23*

Time	Stimulated Area	Ocular Reactions	Other Reactions
11:43	Left frontal lobe............ (distance of coils, 6 cm.)	Right eye deviated to right and upward; left eye showed no movement	
11:44	Right frontal lobe..........	Each eye deviated first to left and then downward	Head deviated to left; cloni of left foreleg
11:45		Spontaneous conjugate deviation to left	
	Right frontal lobe..........	Brief movement of each eye to left	
11:46	Right and left occipital lobes	Are unexcitable	
	Right frontal lobe..........	Conjugate deviation to left	
11:47	Left frontal lobe............	Each eye deviated to right and upward	
11:48		Each eye deviated inward and rotated	
11:51	Left frontal lobe............	Right eye rotated outward; left eye rotated inward and deviated upward	
11:52	Right frontal lobe..........	Deviation of each eye downward and to left; pupils dilated .	General cloni
	Right frontal lobe..........	Each eye deviated upward and to left; after stimulation ceased they moved downward	
11:53	Right and left occipital lobes	No effect	
11:54	Right frontal lobe..........	Each eye deviated upward and to left followed by vertical nystagmus; finally slow downward deviation	General cloni
11:55	Right frontal lobe..........	Each eye deviated downward and to left; then horizontal undulation while the eyes deviated downward	
	Left frontal lobe............	Right eye deviated upward and to left; left eye showed rotation to left	
11:56	Right frontal lobe..........	Same reaction as at 11:55	

(fig. 5) and on more caudal sections to the dorsal portion of the circumference of the central gray matter. The lesion extended caudally to the left inferior colliculus and cranially to the medial nucleus of the right optic thalamus.

These experiments showed that stimulation of the frontal lobes may still yield vertical ocular movements in either direction after destruction of the tectum of the anterior quadrigeminal bodies. The excitability of the oculogyric centers in the occipital lobe was distinctly diminished or lost for ocular movements in either direction after such operations.

In interpreting these findings one has to bear in mind that it is necessary to push the occipital lobes lateralward in order to completely

destroy the tectal region. A certain degree of impairment of the excitability of the occipital lobes seems, therefore, hardly avoidable. In some experiments, at least, rudimentary vertical ocular movements could be elicited by stimulation of the occipital lobes after destruction of the tectum; this was taken to indicate that impulses from the occipital lobes to the subcortical mechanisms for vertical ocular movements do not take their way exclusively over the anterior quadrigeminal bodies but that conduction outside of this ganglion is also possible. This conclusion is corroborated by the fact that centrifugal pathways from the
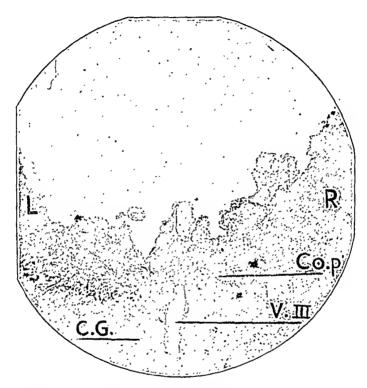
Fig. 5 (cat 23).—Destruction of the tectum of the anterior quadrigeminal body. *C. G.* indicates the central gray matter; *Co. p.*, the posterior commissure; *V. III*, the third ventricle; *L*, the left side, and *R*, the right side.

occipital lobe (the posterior lateral gyrus) could be traced by the degeneration method not only to the pretectal areas and the stratum opticum of the superior colliculus but also to the basis pedunculi, reaching the pontile nuclei (Barris [23]). As for impulses from the frontal lobe, it can be stated with certainty that they can reach the subcortical centers for upward and downward movements of the eyeballs independently of the tectum. Thus the loss of voluntary vertical ocular movements, which originate in the frontal lobe, in cases of lesions in the neighbor-

23. Barris, R. W.: J. Comp. Neurol. **63**:353, 1936.

hood of the aqueduct of Sylvius cannot be ascribed to the destruction of the systems taking their way over the tectum.

The question as to which special group of cells in the region of the central gray matter or its immediate vicinity is concerned in the cortico-fugal impulses for vertical ocular movements to the nuclei for the ocular muscles requires further studies. Some of our observations direct attention to the nuclei in the region of the posterior commissure, the nucleus of Darkschewitsch and particularly the nucleus interstitialis of Cajal, where descending fibers of the posterior longitudinal fasciculus originate (Muskens[24] and van der Schueren[25]). We mentioned the nearness of the stimulation points to the light reflex arc of the pupil and the fact that downward movements were elicited with threshold currents by stimulation closely dorsal to the oculomotor nucleus. The characteristics would correspond to the location of the nuclei of the posterior commissure. Further experiments, however, seem necessary to show on which groups of cells the fibers that carry corticofugal impulses for vertical ocular movements synapse. As for the pathways by which these corticofugal impulses reach the region of the mesencephalic central gray matter, this much can be said: The globus pallidus and its efferent systems are dispensable for such conduction, since we obtained vertical ocular movements in either direction on cortical stimulation after transection of the pallidofugal fibers.[9]

SUMMARY

1. Stimulation of the central gray matter around the aqueduct of Sylvius below the cranial part of the anterior quadrigeminal body may yield constriction or dilatation of the pupil according to whether the pathway of the light reflex or the descending vegetative tracts originating in the hypothalamus are stimulated. Lesions of the central gray matter in this level produce, at most, transitory disturbances of the light reflex; usually they have a short-lived stimulating effect on the sphincter of the pupil, which shows a tonic type of reaction—retardation of its relaxation after cessation of constrictor impulses or on application of dilator impulses.

2. One obtains conjugate downward movements of the eyeballs by stimulation of the central gray matter below the posterior commissure or behind this level and upward movements by stimulation in the posterior part of the optic thalamus or in the medial part of the tectum of the anterior quadrigeminal body. The downward movement is often

24. Muskens, L. J. J.: Brain **36**:352, 1914.

25. van der Schueren: Névraxe **13**:183, 1912.

associated with downward movement of the head in the same direction. The system innervating downward movements lies in the vicinity of the pupillary light reflex arc and the descending hypothalamic pupillodilator tracts; it lies close and dorsal to the stimulation point for convergent movements. Destruction of the central gray matter in the cranial part of the mesencephalon and the pretectal region induces loss of upward and downward movements on cortical stimulation. A small lesion of this region may be followed by various partial disturbances of vertical ocular movements (loss of conjugate movements in one direction only, the appearance of skew deviation on cortical stimulation or loss of vertical movements on stimulation of certain cortical areas, e. g., the occipital lobes only).

3. The tectum mesencephali is not an indispensable part of the system which transmits impulses for vertical ocular movements from the frontal lobes to the nuclei for the ocular muscles. Paralysis of vertical conjugate movements in diseases of the dorsal part of the mesencephalon with intact nuclei for the ocular muscles should therefore be regarded as a local symptom of a lesion of the region around the aqueduct of Sylvius or of closely adjoining systems (the nuclei in the region of the posterior commissure?) but not as a local symptom of a lesion of the tectum.

ABSTRACT OF DISCUSSION

Dr. Clifford B. Walker, Los Angeles: The ophthalmologist as well as the neurosurgeon appreciates most intensely the work of Dr. Spiegel and Dr. Scala on the central connections and pathways of the ocular nerves. In this difficult and intricate field of investigation the findings of Dr. Spiegel and his school deserve the greatest praise. Dr. Spiegel's opening remark, "The region around the aqueduct of Sylvius is of special interest from an ophthalmologic point of view," is certainly true.

Among the numerous ocular disturbances that may come from injuries in the mesencephalic region to the nuclei or pathway of certain nerves, particularly those having control of the oblique muscles, I believe it is possible to include a common type of separated retina.

The following facts and arguments may be considered in this connection:

1. Retinal tears occur so commonly in the upper temporal quadrant in the neighborhood of the attachment of the superior oblique muscle that this is regularly the first region to be investigated by me in routine examination.

2. In over two hundred cases separation of the retina has occurred in only one eye at the same time and from the same trauma. Although double or simultaneous separation does occur, it is decidedly uncommon.

3. The upper portion of the retina is phylogenically older and stronger than the lower portion, yet it is at a mechanical disadvantage

as compared with the lower portion by at least inertia due to the weight of the vitreous (which may weigh over 7 Gm. in myopic eyes). This is because the uppermost portion of the retina, especially if the eye has become softened, must contend with the sag of the vitreous, which tends to produce an area of low pressure between the upper portion of the vitreous and the sclera, while below the vitreous is pressing the retina to the choroid by its weight, causing an area of higher pressure.

4. The superior oblique muscle is attached in the area of low pressure at the top of the eye at the point of termination of the ocular motion of looking downward and outward, and the tendon of this muscle pulls almost at right angles to this spot on the globe on one eye at a time—the right eye, for instance, in looking downward and to the right. A right-handed person has his eyes in this position in many instances at the moment of an accidental jolt that brings on separation. On the other hand, if a person (an actual case is considered) sitting on the right side of an automobile is thrown to the roof by a dip in the road, he very likely will be looking down and to the left for the best place to land. On the way up probably he is looking for a spot without handles and glass on which to fall. He looks down to his left. This direction of gaze may be present on the way down also (if he is not too dazed by bumping the head above) until the spine and pelvis rebound on the seat. My theory is that the fourth nerves receive then a special stimulus which is either direct or transmitted to the mesencephalic decussation in the roof of the fourth ventricle or the Sylvian aqueduct. This extra stimulus may affect each fourth nerve alike, but only one superior oblique muscle—in this case the right superior oblique muscle is already tensely pulling at an angle of about 60 degrees and elevating the sclera when the extra stimulus comes. The other superior oblique muscle is at the same time relaxed. The superior oblique muscle, the longest (60 mm.) ocular muscle, is able to act in this manner because it still has contracting ability to jerk the sclera while the eyeball is held in the position of maximum disadvantage, since its long (20 mm.) tendon is then shortened only about one-half (in the pretrochlear portion) as its attachment comes more directly under the pulley. The shorter the contact arc (5 mm. or less) of the superior oblique muscle (the sagittal type of Fuchs) the more pronounced this action may be. Also, the softer and more weighty the eye (as in myopia) the more effectively can the superior oblique muscle act in the manner just described. The other ocular muscles do not possess to the same extent this mechanical disadvantage making them liable to injury.

It is in the dorsal portion of the mesencephalon (in the wall of the aqueduct, extending to the fourth ventricle), where the fourth nerve decussates more fully than does the optic nerve anteriorly, that it is in a position to receive a stimulus from end thrust impact along the spinal column. From this decussation the fourth nerve winds a long course around both the cerebral and the cerebellar peduncle to reach the orbital fissure. A longitudinal spinal impact may be given lateral components by these peduncles so as to jerk on the anchorage produced by the decussation, which is embedded in a fluid retaining wall somewhat analogous to the chiasmal arrangement of the optic nerve under the

third ventricle anteriorly. Not only can the chiasm be stimulated by certain impacts but it can even be torn sagittally by them.

Dr. W. Lancaster, Boston: I should like to ask Dr. Walker what makes him think that the vitreous weighs 7 Gm.

Dr. Clifford B. Walker, Los Angeles: That is an approximate estimate. When I say that the disadvantage due to weight is 7 Gm. I mean that the weight on the lower portion of the retina is helpfully pressing, while that on the upper portion of the retina is not so helpful and by its inertia contributes to the pressure on the lower portion in case of an upward jerk on the sclera.

Dr. Walter Lancaster, Boston: The whole eyeball is not supposed to weigh 7 Gm.

Dr. Clifford B. Walker, Los Angeles: Its weight has been estimated as between 7 and 8 Gm. If the sclera is removed the weight will be less. Yet so many of the patients with this condition are myopic that some allowance toward the larger number seems fair. But if one fills this ball with water one will find that a sphere 24 mm. in diameter has a weight of about 7 Gm., and the eye has a specific gravity a little over unity.

Dr. Norman P. Scala, Washington, D. C.: I do not know that I can add any more to what has already been said. I wish, however, to thank Dr. Walker for his generous discussion of the paper. Dr. Spiegel and I were of the opinion that supranuclear centers existed for vertical conjugate ocular movements, and we think we have proved that such centers do exist. Paralysis of voluntary vertical conjugate ocular movements should not be considered as a local symptom of a lesion of the tectum mesencephali but as a local symptom of a lesion of the region around the aqueduct of Sylvius or some neighboring or adjoining systems, probably nuclei in the region of the posterior commissure.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

ORBITAL ABSCESSES

RICHARD C. GAMBLE, M.D.

CHICAGO

Orbital abscess is frequently used in its broadest sense to include all types of acute inflammation of the walls and soft tissues of the orbit. Such usage has the virtue of brevity and is perhaps proper in such a review as this. However, in actual clinical work it is of the greatest practical importance to differentiate between a true abscess in the retrobulbar space and a disease limited to the rim or wall of the orbit, such as periostitis, osteomyelitis or subperiosteal abscess. All these conditions may produce the characteristic signs of acute swelling of the lid, proptosis, deep orbital pain and fever. In addition, simple congestion of the orbital veins due to swelling of the mucous membrane in the ethmoid sinuses, as well as extensive spreading thrombophlebitis of the orbital venous system, may produce the same characteristic signs.

Ophthalmologists of fifty or seventy-five years ago were aware of these differences in pathologic anatomy probably just as well as are ophthalmologists of today, but their routine practice of making immediate exploratory incisions into the soft parts of the orbit indicates that they did not fully appreciate the practical importance of making these differentiations. Of course, the one thing which has been of the greatest help to the ophthalmologist in understanding orbital inflammations is the appreciation of the fact that disease of the accessory nasal sinuses is their most frequent cause. Stellwag von Carion[1] in 1868 made no mention whatever of sinus disease in this regard. However, many of the early writers mentioned exposure to cold as a cause of orbital abscess. Fuchs[2] in 1893 stated that retrobulbar phlegmon may arise from periostitis of the wall of the orbit adjacent to an infected nasal sinus. During recent years the sinusal origin of orbital infections has become more and more appreciated. Birch-Hirschfeld[3] expressed the opinion

1. Stellwag von Carion, C.: Treatise on Diseases of the Eye, translated and edited by C. E. Hackley and D. B. St. John Roosa, Baltimore, William Wood & Company, 1868.

2. Fuchs, E.: Textbook of Ophthalmology, ed. 2, translated by A. Duane, New York, D. Appleton & Company, 1892, p. 584.

3. Birch-Hirschfeld: Clinical Course and Therapy of Orbital Phlegmons, Auerbach Jubilee Volume, 1935, p. 84.

that in 60 per cent of cases orbital cellulitis is caused by disease of the nasal sinuses. In his series of six hundred and eighty-four cases in which it was due to sinus disease, the frontal sinus was diseased in 28.8 per cent, the antrum in 21.8 per cent, the ethmoid sinus in 20.5 per cent and the sphenoid sinus in 6.1 per cent. Several sinuses were affected in 14.7 per cent. In a recent report he condemned exploratory puncture into the soft parts of the orbit because it may carry infection into uninvolved tissue and advised making an incision down to the rim of the orbit, followed by elevation of the periosteum until the site of the infection is reached. These are truly words of wisdom from one who has studied the problem for many years. This type of surgical treatment is indicated chiefly in the cases of orbital inflammation due to sinus disease and only in certain stages in such cases. There can be, of course, no routine treatment applicable in all cases at any stage of the development of the inflammation.

ETIOLOGY

The etiologic factors of orbital inflammation are as follows:

1. Extension of infection from adjacent tissues, such as the accessory nasal sinuses, the teeth and the lacrimal sac, and infections of the lid, including erysipelas.

2. Trauma—penetrating and nonpenetrating injuries, including operations on adjacent structures.

3. Bacteremia.

The importance of the nasal sinuses in orbital inflammation is obvious when one considers the frequency with which they become diseased and, further, considers the fact that only a thin layer of bone and two layers of periosteum separate the sinuses from the orbit. There may even be dehiscences in the bone, and arteries, veins and nerves extend through these separating partitions. There is as yet no proof that the lymphatics of the sinuses communicate with those of the orbit, but they may do so. The superior ophthalmic vein communicates with the ethmoid vein; occasionally the inferior ophthalmic vein does likewise. Batson [4] stated that he did not consider the arteries or lymphatics to be important channels for the spread of infection from the nose to the orbit, but that he did consider the veins to be the important route. He also found that when an opaque substance was injected into the anterior facial vein it spread to the superior ophthalmic vein and many of its tributaries. Martin Cohen [5] also stated the belief that the veins are the

4. Batson, O. V.: Relationship of the Eye to the Paranasal Sinuses, Arch. Ophth. **16**:322 (Aug.) 1936.

5. Cohen, Martin: Inflammatory Exophthalmos in Catarrhal Disorders of the Accessory Sinuses, Tr. Sect. Ophth., A. M. A., 1935, p. 209.

important factor. The periorbita is rather loosely attached to the bone except around the rim of the orbit, at the apex and along the various sutures. It can therefore be easily elevated from the bone by collection of pus, with the production of a subperiosteal abscess.

The ethmoid sinus is present at birth, and infection in it can give rise to orbital inflammation at any age. This is the most frequent cause of orbital disease in infants and young children. The antrum is also present at birth but is relatively higher and more mesial than in later years. In infants it is usually diseased if the ethmoid cells are diseased and affects the orbit in much the same manner. The frontal sinuses are not developed at birth and do not become a factor in orbital disease until about the seventh or eighth year. From then on they become an exceedingly important factor. Intracranial complications occur more frequently in cases of orbital disease resulting from infection of the frontal sinus than in the cases of orbital disease due to ethmoiditis. It is probably a fact that whichever sinus is most rapidly developing at a given age is most likely to cause orbital inflammation if it becomes acutely diseased. Thus, one finds disease of the ethmoid sinus and antrum to be the common cause of orbital inflammation in infants and children up to the age of 9 or 10. Then the frontal sinus becomes more important. In adults these three sinuses are about of equal importance.

Infection in the tooth buds furnishes another example of the tendency for inflammation to arise in rapidly developing tissues. This occurs most often in nursing infants and may arise as a result of trauma to the mucous membrane of the gums, the infection spreading upward into the tooth buds and then to the floor of the orbit, as has been suggested by Marx.[6] A different origin for this type of infection was suggested by van Gilse,[7] who stated that the bony case of the maxillary sinus is not completely closed in an infant, so that infection may pass from the nose to the antrum and then to the tooth buds, with resulting osteomyelitis of the superior maxilla.

Infection in the lacrimal sac may spread to the orbit either spontaneously or as a result of operations. This happens less often than one would expect, probably because the deep fascia covering the sac gives excellent protection. I have seen it occur as a result of too liberal use of trichloro-acetic acid and also following curettage of the interior of the sac. John Green[8] has reported a case of orbital abscess which followed hordeolum.

6. Marx, E.: Eye Symptoms Due to Osteomyelitis of Superior Maxilla in Infants, Brit. J. Ophth. **6**:25 (Jan.) 1922.

7. van Gilse, P. H.: Die pathologische Bedeutung der Nebenhohlen der Nase bei kleiner Kinder, Maandschr. v. kindergeneesk. **3**:431, 1934.

8. Green, John: Management of Orbital Infections, Am. J. Ophth. **14**:196 (March) 1931.

Erysipelas must have been a common cause of orbital disease in years past, judging from the prominent place given to it by the early writers. One early textbook makes the statement that in many cases of orbital cellulitis of apparently unknown origin the condition was probably due to slight attacks of erysipelas of which the patient was not aware. The physicians of those days must have been erysipelas conscious as a result of their experiences in military surgery. Certainly it is not a common cause of orbital disease at the present time. However, Bellows [9] found that among one hundred and thirty patients with facial erysipelas, six had orbital cellulitis. He expressed the belief that it develops by thrombophlebitis and leads to multiple small abscesses which may become confluent. Incision may be inadequate, and in one case evisceration of the orbit was done because there were threatening signs of meningitis.

Trauma may cause orbital inflammation without any penetrating injury being present. This ordinarily occurs as a contusion from injury from a blunt instrument and results in periostitis of the rim of the orbit. However, this may spread deeper to the wall of the orbit and cause proptosis. Penetrating injuries from foreign bodies of various types are more common and are more serious. Organic materials, such as splinters of wood, often harbor bacteria and are therefore more prone to cause suppuration than glass or metallic substances. Surgical traumas, such as the extraction of infected teeth and operations on the nasal sinuses, at times result in malignant orbital infection, frequently of a thrombophlebitic type.

Bacteremia causes a considerable proportion of orbital infections. As a rule, the process begins as periostitis, but because the patient is already seriously ill it rapidly spreads into the retrobulbar tissues and forms a true orbital abscess. This occurs most frequently as a complication in the exanthematous diseases, especially scarlet fever and smallpox and, to a lesser extent, measles. It occurs less often in pneumonia and puerperal sepsis. In the exanthematous diseases it is often difficult to determine whether the orbital disease is due to bacteremia or is a result of the purulent rhinitis which often is present. In cases of orbital abscess complicating scarlet fever the infective agent is ordinarily the streptococcus; in cases of smallpox and measles the orbital abscess is due to secondary invaders. In general, the streptococcus, the hemolytic staphylococcus and the pneumococcus are the organisms found in the abscess. Beigelman [10] reported the case of a patient who had an actino-

9. Bellows, John: Ocular Complications of Erysipelas, Arch. Ophth. **11**: 678 (April) 1934.

10. Beigelman, M.: Actinomycosis of the Orbit, Arch. Ophth. **10**:664 (Nov.) 1933.

mycotic infection in the orbit. The process lasted for two years, and death occurred from low grade meningitis and abscess of the brain. Adams [11] found reports in the literature of eight cases of infections in the sinuses due to Aspergillus and added a case of his own. Eight patients were women, and only one was a man. Proptosis was an early sign, and there was a profuse nasal discharge.

PATHOLOGICO-ANATOMIC FEATURES

It is not always possible to determine the pathologico-anatomic features from the clinical appearance, but careful consideration of the etiology will greatly aid one's judgment in this respect. When disease of the nasal sinuses is the cause, the nasal wall of the orbit is usually involved. In children this is true in regard to the frontal and maxillary sinuses, as well as the ethmoid sinus, because they have not extended laterally very far. In older patients the upper and lower quadrants are affected in disease of the frontal and the maxillary sinuses, respectively. In many of the cases of milder inflammation there is only venous congestion in the orbit for a few days, due to compression of the ethmoid vein by the swollen mucous membrane in the ethmoid sinus. Edema of the upper lid and slight proptosis may be the only signs. This type is common in young children. If the sinus disease becomes more extensive it may cause some osteomyelitis of the nasal wall of the orbit, and the infection may cause a subperiosteal abscess. This leads to greater swelling of the lid and greater proptosis and usually to fixation of the eyeball. This abscess may penetrate the periosteum and cause an abscess in the soft parts of the orbit, or it may extend back to the apex of the orbit and cause meningitis or thrombosis of the cavernous sinus, but it usually dissects forward and appears as a subcutaneous induration in the lids, usually above or below the internal canthal ligament. In these cases the true contents of the orbit are spared, and recovery is complete and prompt as soon as the subperiosteal abscess is drained. When such an abscess points in the conjunctiva, either the fornix or on the eyeball, it means that the pus has dissected through the soft tissues, and the orbital structures may be permanently damaged. In some cases the subperiosteal abscess drains back into the nose, and recovery is spontaneous. This is true of the cases of orbital abscess due to sinus disease, not of cases of orbital abscess of the other types.

Many times the process starts as acute periostitis. This is true especially of those cases in which the orbital abscess is due to nonpenetrating injury of the orbital rim and of those cases in which it is

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11. Adams, N. F., Jr.: Infection Involving the Ethmoid, Maxillary and Sphenoid Sinuses and the Orbit Due to Aspergillus Fumigatus, Arch. Surg. **26:** 999 (June) 1933.

associated with bacteremia. In the cases in which it is due to nonpenetrating trauma it is usually not severe. In the cases in which it is due to penetration of foreign bodies it varies considerably in severity according to the type of infection carried in. Splinters of wood usually cause more severe inflammations than other injuries and, incidentally, are more likely to cause tetanus. The type of orbital abscess associated with bacteremia is very severe and unless promptly treated usually extends into the orbit and causes an abscess of the soft parts, frequently resulting in damage to the optic nerve or the nerves to the muscles.

Osteomyelitis of the superior maxilla occurs most frequently in infants and children and appears to arise spontaneously. The acute form is most common. The entire cheek is swollen and indurated and is often dark. In 10 per cent of these cases the floor of the orbit is involved sufficiently to cause proptosis. There is considerable swelling of the lid in practically all cases. The mortality in cases of this type is about 25 per cent. The entire maxilla is usually involved, and discharging fistulas may appear along the lower rim of the orbit, through the palate or into the canine fossa. Ultimately a chronic stage is reached in which the acute swelling subsides but the fistulas remain. Rough bone denuded of periosteum may be felt at the bottom of these fistulas, and spicules of loose bone may be extruded. There is also a type of osteomyelitis in which the infection is of low grade throughout its course. The fistulas appear in the lower lid and cause adherent depressed scars. There may also be discharging fistulas along the alveolar process in association with very carious teeth. This type of infection resembles the chronic inflammation of the superior maxilla due to tuberculosis or syphilis.

SIGNS

In addition to the general evidences of infection, fever and leukocytosis (which are not invariably present), the cardinal signs of orbital inflammation are redness and swelling of the lids and conjunctiva, proptosis and fixation of the eyeball. Chemosis occurs when the pus nears the surface. Induration of the subcutaneous tissues results when the abscess has dissected its way forward under the skin.

COMPLICATIONS

The question of complications resulting from orbital inflammation deserves careful consideration. In some cases immediate and radical treatment is essential; in others too much treatment is literally fatal. The important factors are the severity of the condition, the cause, the age of the patient and the status regarding other existing complications.

In young children orbital inflammation due to sinus disease is usually not very dangerous if handled in a conservative manner. Many of the

patients recover spontaneously or with simple treatment. As a rule, even though an abscess forms, it is subperiosteal and readily dissects its way forward and can be evacuated by a simple incision of the skin. The few days required for this to occur gives time for a protective pyogenic membrane to form. Complications in this type of orbital inflammation are not common. If an incision is made too early or if extensive intranasal or external operations on a sinus are done in these cases of acute disease the mortality is very high, especially in patients under 2 years of age, and death occurs more often from pneumonia, empyema and general sepsis than from meningitis or disease of the cavernous sinus. This is an important fact and is, perhaps, more thoroughly appreciated by the pediatrician than by the ophthalmologist, or the rhinologist. In children 10 years of age or more, disease of the frontal sinus is often the cause. In these patients the condition may be very severe and may frequently result in abscess of the frontal lobe of the brain or meningitis. In most cases of orbital inflammation there is probably more or less infective thrombophlebitis. In those cases in which an abscess forms in the orbital contents this condition is probably extensive. Occasionally it spreads to the cavernous sinus, but not nearly as often as one would expect. Retrograde extension of infective thrombophlebitis into the optic nerve and eyeball probably accounts for the neuritis and the later atrophy of the optic nerve which are often seen, and also for the rare occurrence of massive exudative choroiditis, which may be found associated with severe involvement. A slight congestion of the retinal veins is commonly seen and is of no great importance. Ulceration of the cornea may occur as a result of exposure resulting from the exophthalmos and is perhaps aided by a certain degree of damage to the ciliary nerves due to pressure within the orbit. Also, corneal abrasions may be caused by gauze dressings and drains. The corneas of adults are less resistant to ulceration than are those of children, but the latter are not exempt from ulceration.

Strabismus of a paralytic type sometimes follows orbital abscess, either because the nerve to a muscle has been damaged within the orbit or because the action of a certain muscle is limited by scar tissue. In children it is not unusual to have the onset of impending concomitant squint precipitated simply because for several weeks the eye has been occluded by a dressing or swelling of the lid, or both. It is wise, therefore, in all cases of orbital abscess in children to occlude the unaffected eye for certain periods after recovery, to prevent suppression of vision.

TREATMENT

The treatment of acute inflammations in the orbit is to a great extent determined by what one knows concerning the etiology and the pathologico-anatomic features. Immediate routine exploratory incision

through the conjunctival fornix into the soft retrobulbar tissues in all cases of acute proptosis is a thing of the past. There are several reasons for condemning this practice: There may be no pus to evacuate; the pus may be entirely subperiosteal (and in that event the infection would gain access to the retrobulbar tissues as a result of the operation), and in many cases it is safer to allow time for the abscess to become walled off. On the other hand, the danger of permitting a true orbital abscess to continue undrained is considerable. Mention has previously been made of the fact that Birch-Hirschfeld condemned exploratory puncture and advocated a subperiosteal approach. Hall [12] reported twenty-three cases of orbital infection in which there were four deaths. He divided his cases into two groups, thoses in which the condition was due to external infection such as furuncles, erysipelas and trauma and those in which it was due to sinus infection. All the deaths occurred in the first group. The patients required radical treatment. He expressed the belief that patients in the second group should have more conservative treatment. A number of writers have pointed out that in a high percentage of cases of orbital infection due to sinus disease the patient recovers safely following the application of astringents to the nasal mucous membrane and nasal suction or, at most, incision through the skin to evacuate a subperiosteal abscess after it has neared the surface. Among these writers are Moller,[13] Ballenger,[14] Gamble [15] and Greenfield.[16] Greenfield in cases of orbital infection due to sinus disease uses nasal astringents and suction, and performs incision and drainage only if the eye becomes fixed and induration is present. In two cases he was able to demonstrate by roentgen examination that the ethmoid labyrinth was dilated; that is, the distance between the nasal septum and the orbital plate was greater on the affected side than it was on the normal side.

The results obtained by those advocating this type of treatment have been good. A number of other writers who have favored external operations on the sinuses in similar cases, such as Porter [17] and Phelps,[18]

12. Hall, J. S.: Orbital Infections, J. Laryng. & Otol. **50**:690 (Sept.) 1935.

13. Moller, J.: Ueber orbital Phlegmone, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **25**:458, 1935.

14. Ballenger, H. C.: Orbital Cellulitis and Abscess Secondary to Sinusitis, Illinois M. J. **61**:128 (Feb.) 1932.

15. Gamble, R. C.: Acute Inflammations of the Orbit in Children, Arch. Ophth. **10**:483 (Oct.) 1933.

16. Greenfield, S. D.: Acute Sinusitis in Children Associated with Orbital Complications: The Conservative Treatment, Laryngoscope **44**:683 (Sept.) 1934.

17. Porter, C. T.: The Etiology and Treatment of Orbital Infections, Ann. Otol., Rhin. & Laryng. **41**:1136 (Dec.) 1932.

18. Phelps, K. A.: Cellulitis of Orbit in Infants and Children, Ann. Otol., Rhin. & Laryng. **33**:1391 (Dec.) 1924.

have reported results which are perhaps equally good. It may be said, however, that this type of treatment does not yield as good results in young children as it does in older patients.

There have been a number of cases of exceptionally severe orbital infection which has required still more radical surgery. Eagleton[19] described infections in the retrobulbar space of thrombophlebitic nature which led to sudden blindness from arterial spasm and were part of the picture of thrombosis of the cavernous sinus. He advised immediate drainage of such abscesses by a latero-orbitocranial approach which involves rather extensive removal of bone. Martin Cohen[5] performed a Krönlein operation in two cases of long-lasting severe exophthalmos due to chronic sinus disease, and in one case he performed a Naffziger operation.

In regard to osteomyelitis of the superior maxilla, all writers have emphasized the serious nature of the disease and the difficulties in treating it. Posey[20] mentioned the need for multiple operations, including incision for drainage and later removal of sequestrated bone and dead teeth. Custodis[21] advised early and extensive surgical treatment. He reported 5 cases; 4 of the patients died. Marx[6] refrained from even making an incision for drainage in this type of case. The process is so diffuse and the induration so marked that even multiple incisions do not give adequate drainage. Pneumonia or general sepsis frequently follows such surgical intervention.

19. Eagleton, W. P.: Exophthalmos from Surgical Diseases, Arch. Ophth. **14**:1 (July) 1935.

20. Posey, W. C.: Orbital Cellulitis from Disease of the Superior Maxilla in Children, Tr. Sect. Ophth., A. M. A., 1912, p. 229.

21. Custodis, E.: Inflammatory Diseases of the Orbit in Osteomyelitis of the Superior Maxilla in Infants, Klin. Monatsbl. f. Augenh. **87**:631 (Nov.) 1931.

Correspondence

AN ANACHRONISTIC STATEMENT

To the Editor:—In a paper entitled "Secondary Cataract with Particular Reference to Transparent Globular Bodies," by Drs. Alfred Cowan and Wilfred E. Fry, published in the July issue (ARCH. OPHTH. 18: 12, 1937), on page 22 there is the following statement referring to a paper by Elschnig: "In the report of neither case is there an accompanying description of the results of clinical or slit lamp examination." On page 12 the reference to Elschnig's paper is given (*Klin. Monatsbl. f. Augenh.* 49: 444, 1911).

It seems to me that the statement is rather anachronistic, considering the fact that Alvar Gullstrand published his illumination method for the first time in 1911 (Demonstration of Nernstslitlamp, Ber. ü. d. Versamml. d. ophth. gesellsch. 37: 374, 1911), and owing to the efforts of O. Henker, of the Zeiss Works, the Gullstrand lamp was combined with the corneal microscope in 1916. So far as I remember, the pioneer publications date from 1918, and therefore Elschnig could not have given a description of the results of slit lamp examination in 1911.

ANDREW RADOS, M.D., Newark, N. J.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

SOCIETY NEWS

National Society for the Prevention of Blindness.—The annual conference of the National Society for the Prevention of Blindness was held in New York on October 6 to 8. This year the opportunity was given to participate in the American Public Health Association's annual convention. Discussions and papers were presented on the following subjects:

"Saving Sight Through Public Action," by Dr. William F. Snow.

"Syphilis of the Eye as a Factor in Industry," by Dr. Park Lewis.

"Possibilities for Further State-Wide Development of Sight Conservation Through Cooperation of Interested Agencies" (informal discussion).

"The Vision of the School Child," by Francia Baird Crocker, R. N.

International Organization Against Trachoma.—There will be sessions of the International Organization Against Trachoma during the period of the Fifteenth International Congress of Ophthalmology, to be held in Cairo, Egypt, from Dec. 8 to 14, 1937. On December 9 there will be a meeting of the present executive committee, which will be followed by a plenary meeting of the delegates from the national ophthalmologic societies together with the subscribing members of the International Organization Against Trachoma. The names of such delegates should be sent to the president of the International Organization Against Trachoma, 33 Welbeck Street, London, W. I., at least thirty days before the opening of the congress. The following program of the scientific sessions will be presented:

Introductory address by the president, Dr. MacCallan.

"The Microbiologic Etiology of Trachoma," by Dr. Phillips Thygeson, United States of America; Dr. Grüter, Germany; Dr. Oguchi, Japan; Drs. Cuénod and Nataf, Tunis; Dr. Poleff, Morocco, and Dr. Rötth, Hungary.

"The Pathology of Trachoma," by Dr. Wilson, Egypt; Dr. Michail, Roumania; Dr. Mulock-Houwer, Netherland Indies, and Dr. Pascheff, Bulgaria.

"The Treatment of Trachoma," by Dr. Sobhy Bey, Egypt, and Dr. Shimkin, Palestine.

"The Epidemiology of Trachoma in Japan," by Dr. Motegi, Japan.

"Trachoma Among School Children in Buenos Aires," by Dr. Lijó Pavía, Argentina.

"Trachoma in Turkey," by Dr. Ayberk, Turkey.

UNIVERSITY NEWS

Dr. Francis P. Guida, for the past two years resident in ophthalmology at Yale University, has been appointed full time instructor in ophthalmology at the University of Chicago.

Dr. Louis Bothman has been promoted to the position of clinical professor of ophthalmology at the University of Chicago.

Abstracts from Current Literature

Anatomy and Embryology

RELATION BETWEEN THE MUCOPROTEIN AND THE FRAMEWORK OF THE
VITREOUS. J. GOEDBLOED, Arch. f. Ophth. 137: 131 (April) 1937.

A comparison of the properties of the whole vitreous with those
of the dialyzed fluid of the vitreous reveals a striking congruity. The
properties of the dialyzed fluid of the vitreous are determined by the
specific protein present in that fluid, namely, mucoprotein. Goedbloed
therefore assumes that the actual structure of the vitreous is that of
filaments made up of the residual protein (vitrein) surrounded by films
of mucoprotein. Such a conception would explain why, colloidochemi-
cally, the whole vitreous behaves like mucoprotein.

·P. C. KRONFELD.

CYTOLOGIC CHARACTERISTICS OF THE HUMAN RETINA. Y. UYAMA,
Arch. f. Ophth. 137: 318 (June) 1937.

The horizontal cells are better developed in the human retina than
in the retina of the other mammals. Neurofibrillae form a network
which connects the horizontal cells directly with one another, without
the action of their processes. The crystalloid bodies which Kolmer
described in the horizontal cells are probably cholesterol crystals and
have nothing to do with the mitochondria or other protoplasmic parts
of the cell body. Of the amacrine cells two types can be distinguished,
namely, spherical or elliptic cells with long processes, and smaller, but
more numerous cells which contain neurofibrillae only on one side of
the nucleus. These fibrillae describe conspicuous spirals before emerging
from the cell bodies.

P. C. KRONFELD.

Biochemistry

OBSERVATIONS ON THE REDUCING SUBSTANCES (GLUCOSE) OF THE
AQUEOUS AND VITREOUS HUMORS OF THE EYE. W. M. JAMES
and A. J. SIEFKER, Am. J. Ophth. 19: 975 (Nov.) 1936.

After carrying out experiments on rabbits, James and Siefker arrived
at the following conclusions:

"1. In fasting rabbits under dial-Ciba anesthesia the blood sugar
averages 137 mg. percent, the aqueous sugar 164 mg. percent, and the
vitreous sugar 104 mg. percent.

"2. Changes in the glucose level of the blood are reflected after a
lapse of time in the intraocular fluids. An increase in the glucose con-
tent of the aqueous increases the osmotic pressure in the anterior
chamber, and with the subsequent fall of the blood-sugar level the
accumulation of glucose in the aqueous causes the inflow of fluid into
the anterior chamber with a subsequent rise in intraocular pressure.

The relatively lower osmotic pressure of the vitreous humor facilitates the passage of fluid from the posterior to the anterior segment of the eye.

"3. Eserine increases the rate of the diffusion of glucose into the aqueous of the eye.

"4. Atropine retards the diffusion of glucose into the aqueous humor of the eye.

"5. Irritation of the vascular bed of the uveal tract is associated with wide fluctuations in intraocular pressure. These fluctuations are due to vascular dilatation or constriction and also to alterations of the permeability of the capillary membrane."

W. S. Reese.

Researches Concerning the Chemical Constituents of the Retina, Their Relations to the Retinal Purple and the Theory of the Duality of Vision. M. Verrier and R. Pannier, Bull. Soc. d'opht. de Paris, July 1936, p. 569.

The theory of the duality of vision has for a long time dominated the concept of the physiologic nature of vision. All the recent research seems to rest on a small number of morphologic facts, not generally authenticated, while the facts contrary to the theory have been neglected. Instead of the dual morphologic nature of the visual cells it is convenient to substitute a notion of a polymorphic assembly of these elements. This polymorphism is found by a comparative study of the retina of man and of the vertebrates. The authors have found that the reparation of the visual purple is independent of the form of the visual cells. A chemical study of the visual cells would apparently bring forth many facts of interest concerning the matter. Wald's work, which identified the retinal purple as vitamin A, is most important. This work was done on the frog and some of the fishes. Brunner, Baroni and Kleinau reported the coexistence of B carotene and the rods, but they limited their study to the eyes of beef and swine without establishing a comparison with the retinas unprovided with typical rods. Finally and more recently, Euler, Hellström and Adler placed the localization of the flavine in the retina at the level of epithelial pigment. Their observations were made on the rabbit, the rat, the ox and man.

In the present article the authors report studies on solution of taurocholate, vitamin A, carotene albumin, melanin, flavine and other substances. They conclude that the retinas of various species contain the same constituents in varying proportions.

L. L. Mayer.

Cornea and Sclera

Keratoconjunctivitis with Adenitis in Calcutta. S. Sanyal, Am. J. Ophth. 19: 982 (Nov.) 1936.

A form of keratoconjunctivitis is described, characterized chiefly by small hyperemic areas on the conjunctival surface of the upper lid, in which are one or two yellowish pinpoint-sized areas. The symptoms, course, treatment and histopathology are given, and the following summary is made:

"A form of keratoconjunctivitis with adenitis is described which in its protean manifestations resembles many known diseases of the conjunctiva and cornea with which it has been confused but which bacteriologically is shown to be the same disease.

"Pannus in a case of conjunctivitis in man is regarded by most authorities as the strongest evidence that the disease is trachoma. In guinea pigs and rabbits a form of conjunctivitis with pannus occurs naturally; it has also been experimentally reproduced. This pannus differs from the pannus of hypersensitiveness in being unable to produce curtain pannus. The disappearance of the bacteria by small tarsectomies, whereas clinically the granules persist, and the production of greater amounts of fibrous tissue raise an interesting point in trachoma, that probably many cases are spontaneously cleared of bacteria although clinically the signs persist.

"It will be seen that the corneal affections are different from the epidemic superficial keratitis which has been described previously. The monocular phase formed the subject matter of another paper."

W. S. Reese.

The Results of Corneal Transplantation. J. W. Tudor Thomas, Brit. M. J. 1: 114 (Jan. 16) 1937.

The author begins by stating that successful results in corneal transplantation can now be obtained, but conclusions as to the satisfactoriness of the procedure will depend on the publication of detailed reports of all the cases in which this treatment is used.

The cases should be classified according to a definite scheme, and a uniform classification is suggested. This scheme consists in classifying the eyes into three groups: group 1, suitable eyes in favorable patients; group 2, suitable eyes in unfavorable patients and group 3, unfavorable eyes. The results in each group should be given separately. Success depends on the degree of transparency of the graft and the absence of an opaque membrane immediately posterior to it. The results should be analyzed on the basis of the transparency of the graft after a definite period rather than on that of visual acuity.

A summary is given of the results of Elschnig, Filatov, Castroviejo, and Tudor Thomas, and the following conclusions are reached: In order to insure the union of the graft the cutting of the graft shelving, Tudor Thomas' original suggestion, is essential. On the whole, it may be stated that the results in favorable cases were 50 per cent and those in unfavorable cases 38 per cent, and from Tudor Thomas' own figures the chances of obtaining a graft that can be described as transparent are 27 per cent.

The author is convinced that corneal transplantation can now be recorded as a practical procedure, though much is still to be done to make the treatment more effective and to improve the operative technic.

Arnold Knapp.

Unusual Case of Xanthomatosis Corneae. A. Szász, Arch. f. Augenh. 110: 373, 1937.

The right eye of a 43 year old woman showed moderately advanced trachomatous pannus. Several injections of trachoma vaccine were

given. Four years later the patient returned with a yellowish mass over the upper third of the cornea. Histologic examination showed simple granulation tissue. Four years later the patient returned, and at that time a yellowish tumor covered the entire cornea. The tumor was removed and on examination was found to consist of granulation tissue with a varying cellular structure. Some foreign body giant cells were present, and there were a great many cells which had a foamlike structure, due to the presence of droplets of fat in the cells. These cells were xanthomatous. Shortly thereafter the globe was enucleated and examined histologically. Xanthomatous cells were at this time noted throughout the cornea. The author concludes that fats were deposited in the affected tissues as a result of disturbed local metabolism and not as a result of fatty degeneration of the tissue. The main collection of xanthomatous cells was at some distance from the infiltrated region.

<div align="right">F. H. Adler.</div>

Experimental Pathology

CHANGES IN THE CORNEAL NERVES AFTER EXTIRPATION OF THE GASSERIAN GANGLION IN RABBITS. K. REISER, Arch. f. Augenh. 110: 253, 1937.

The gasserian ganglion was extirpated on one side in a series of rabbits, and after various periods of time the rabbits were killed and both eyes enucleated. The corneas of these eyes were sectioned and examined for degenerative nerve fibers. None of the rabbits lived longer than seventy hours, but Reiser points out that this period is sufficient for all the signs of degeneration in the nerve elements to appear. He found that after thirty hours the first evidences of degeneration are seen in the large nerve bundles of the cornea. There occur nodular swelling of the nerve fibers and clumping together of many nerve fibers into broad bands. Scattered through these bands are round bodies, apparently the result of fibrillolysis. The more peripheral sections of the corneal nerves showed fewer changes. In the preterminal network there were practically no pathologic changes. The nervous terminal reticulum seemed to remain free from any changes and did not degenerate. This, Reiser believes, is further proof of the syncytial nature of the peripheral nerve elements.

<div align="right">F. H. Adler.</div>

General

HEREDITY OF SJÖGREN'S DISEASE ASSOCIATED WITH KERATOCONJUNCTIVITIS SICCA. R. LISCH, Arch. f. Augenh. 110: 357, 1937.

Lisch describes the condition generally referred to as Sjögren's disease in three successive generations. Of thirteen persons examined, eleven showed definite signs of this symptom complex, which consists of dryness of the mouth, nose and throat, difficulty in swallowing dry food and, occasionally, hoarseness. Associated with this occasionally there was absence of tears, with dryness of the conjunctiva. Two patients suffered from filamentary keratitis with diminished corneal sensitivity. In a few of the patients studies of the gastric secretions

showed subacidity. No changes were found in the blood of any of the patients to suggest pernicious anemia as claimed by Grósz. Lisch discusses the possibility of this disease being due to some disturbance in the vitamin metabolism and establishes the familial symptom complex.

F. H. ADLER.

General Diseases

OCULAR CHANGES IN SCHÜLLER-CHRISTIAN-HAND'S DISEASE. C. BEHR, Arch. f. Ophth. **136**: 403 (Feb.) 1937.

Behr reports two cases of the type of lipoidosis which in America is best known as Christian's disease. It consists of the development of circumscribed granulomas with heavy lipoid infiltration in various parts of the body, chiefly in the bones of the skull. Secondarily softening and hemorrhages occur in these granulomas. The outstanding clinical symptoms are maplike defects in the bones of the skull, exophthalmos and diabetes insipidus. The author analyzes the ocular changes which develop in the course of the disease. The exophthalmos may be caused by a lipoid granuloma located either in the bony wall of the orbit or in the sheath of the optic nerve (parenchymatous lipoidosis). The latter form is difficult to diagnose. The diabetes insipidus in Christian's disease is produced by granulomas located in or near the hypophysis, the tuber cinereum or the infundibulum. In one of the cases reported by Behr the patient improved greatly while kept on a diet low in fat. In the other case there was only one orbital lesion, which was removed surgically. No local recurrence and no other lesion developed during the subsequent five years.

P. C. KRONFELD.

Lacrimal Apparatus

CRITICAL STUDY OF THE MEDICOCHIRURGICAL TREATMENT OF DAC- RYOCYSTITIS. R. ARGAÑARAZ, Arch. de oftal. de Buenos Aires **11**: 567 (Oct.) 1936.

Argañaraz gives a historical review of the treatment of dacryocystitis since the time of Hippocrates to the present day. He considers that drainage of the lacrimal sac is the fundamental factor in the cure of chronic dacryocystitis; the better the drainage, the better the result. Dacryocystitis is due not to a nasal obstruction but to the existence of a closed septic cavity. By opening freely the external wall of the sac toward the inner angle of the eye, free communication is established between the conjunctiva and the lacrimal sac, transforming the latter into a conjunctival culdesac and curing the dacryocystitis but not the epiphora.

After a further study of the origin of the epiphora, the relation of the permeability of the lacrimal secretory apparatus to tearing and the cause of dacryocystitis, Argañaraz considers the indications for probing, excision of the lacrimal sac and dacryocystorhinostomy. He describes his procedure of opening the lacrimal sac through the conjunctiva, combined in obstinate cases with puncture of the bony wall with a special trocar, as a substitute for the two latter operations. He considers the endonasal Toti operation the ideal procedure.

C. E. FINLAY.

Lens

INFLUENCE OF PHLORHIZIN AND SODIUM FLUORINE ON THE VITAMIN C CONTENT (CAPACITY OF REDUCING DICHLORPHENOLINDOPHENOL) OF THE LENS. H. K. MÜLLER, Arch. f. Augenh. 110: 321, 1937.

The following experiments were undertaken to determine whether the vitamin C content of the lens would be influenced by poisoning animals with phlorhizin and fluorine.

In a series of ten normal rabbits the vitamin C content of the lens was found to be 26.2 mg. per hundred grams, that of the aqueous 31.2 mg. and that of the adrenal glands 299 mg.

A second series of rabbits of the same race, age and state of nutrition were given 0.05 mg. of phlorhizin per gram of body weight twice daily subcutaneously, over a period of twenty-one days. Occasionally some of the poison was also administered subconjunctivally. At the end of the treatment the animals were killed, and the vitamin C content of the lens, aqueous and adrenal glands was immediately determined. It was found that the vitamin C content had diminished in the lens to an average of 16.5 mg. per hundred grams, that in the aqueous to 21.2 mg. and that in the adrenal glands to 199.5 mg.

A second series of animals were given fluorine, and similar studies were made. These animals showed a slight increase in the vitamin C content of the aqueous, lens and adrenal glands.

Müller discusses the various chemical possibilities by which the changes in the vitamin C content may occur. He concludes that in his studies this was due either to some disturbance in glycolysis or to some influence on the permeability of the aqueous-blood barrier.

F. H. ADLER.

Methods of Examination

A NEW ADAPTOMETER. J. LIJO PAVÍA, Arch. de oftal. de Buenos Aires 11: 341 (June) 1936.

The author refers to a prior paper of his, read in May 1930 before the Argentine Ophthalmologic Society, to a paper of J. Nordman and to one of J. Nordman and P. Payeur, on the adaptation for clinical purposes of Drem's ingenious photometer in common use in cinematographic work. He believes that this device is practical for the following reasons: (1) low cost, (2) facility of adaptation to the eye, (3) rapid working, (4) direct reading of the scale and (5) uniformity of illumination.

C. E. FINLAY.

ENTOPTIC METHODS. E. P. FORTIN, Arch. de oftal. de Buenos Aires 11: 628 (Nov.) 1936.

Fortin describes in detail numerous entoptic findings with modern illuminating apparatus, which permit an accurate study of the affected parts of the eye, furnishing data often superior to those furnished by ophthalmoscopy. The author cannot understand why this method of examination has been so persistently ignored.

C. E. FINLAY.

CLINICAL EXPERIENCES WITH THE NEW ZEISS PROJECTION PERIMETER (AFTER MAGGIORE). H. SERR, Arch. f. Ophth. 136: 477 (Feb.) 1937.

The main advantages of the projection perimeter over the ordinary perimeter are: uniform brightness of the stimulus, irrespective of its place of appearance on the arc of the perimeter, and the absence of distracting visual or acoustic stimuli. Perimetry can be carried out with the projection perimeter more quickly and with less fatigue than with the ordinary perimeter. The brightness of the stimuli can be varied so that perimetry on the semidark-adapted eye is possible. The projection perimeter is also suitable for the detection of small central and paracentral scotomas.

P. C. KRONFELD.

Neurology

MECHANISM OF HEADACHE. T. FAY, Arch. Neurol. & Psychiat. 37: 471 (Feb.) 1937.

Through the application of faradic stimulation traction irritation and heat to the structures within the cranial cavity, with the patient under light anesthesia or with the part under local anesthesia, Fay observed that a sensation of pain similar to localized headache could be produced only when the branches of the meningeal artery are directly involved. Referred pain similar to headache could be elicited by stimulation of the superior sinus from a point approximately underlying the hair line of the forehead to the torcular Herophili, the two lateral and the two sigmoid sinuses. Great pain was experienced from the traction or stimulation of the branches of the venous supply, the tentorium, the inferior straight sinuses and the cavernous sinus and, on one occasion, by stimulation of the right carotid plexus.

Fay noted the following with regard to pain referred to the eyes: Stimulation of the arterial tree near the branches of the circle of Willis produced pain referred deep into the eyes and forehead. Stimulation of the superior sinus produced diffuse pain, regional in character, referred to a point deep in the eyes, especially as the point of stimulation approached the torcular Herophili. Stimulation of the tentorial veins and lateral sinuses produced pain referred to the occipital area and deep behind the eyes. Compression of the jugular vein of the neck, with pressure in the lateral sinus and posterior portion of the superior sinus, produced occipital headache referred deep in the eyes, when the vascular engorgement reached a point of actual gross tumescence of the dural sinuses.

The author believes that his observations show that fibers for pain situated on the large vascular branches of the cranial structures leave the skull, face and head via their vascular structures to descend into the upper part of the thorax by various roots and into the spinal cord through the posterior route mechanism between the seventh cervical and the fifth thoracic segment of the cord.

In summary, Fay reduces the pathogenesis of headache to the common denominator of pain due to stretch of the cerebral vascular tree when extremes of hydration or dehydration are such as to alter

grossly the volume relationships of the cranial cavity and bring about physical stretch on the sheath of the large arteries and venous sinuses. He suggests treating by hydration or dehydration, whichever counteracts the cause of the symptom in the particular instance. He gives his criteria for deciding whether a headache is due to hydration or to dehydration.

R. IRVINE.

OCULAR PARALYSIS FOLLOWING MUMPS. T. HARRISON BUTLER AND A. J. WILSON, Brit. M. J. 1: 752 (April 10) 1937.

This is a report of two cases of neurologic complications of unusual localization after mumps.

Case 1 is that of a boy of 9 years who, three weeks after he had been taken ill with mumps, showed paralysis of the soft palate and of accommodation. There was slight blurring of the edges of the disks. The knee jerk was absent on one side. Examination for diphtheria gave negative results. When seen again a number of years later the eyes were found to be normal. The case is interesting as an example of the picture of postdiphtheritic paralysis of the intrinsic muscles of the eyes caused by the virus of mumps.

Case 2 is that of a boy of 12 years who, a month after an attack of mumps, had difficulty with vision in the left eye and inability to see anything on the right side. The eyegrounds were normal. There were complete paralysis of the left third nerve and paralysis of the soft palate. There was no evidence of diphtheria. A few days later the left external rectus muscle became affected. The superior oblique muscle, however, remained active. A year later there was still paralysis of the third nerve.

The two cases are of interest because of the type of nerves involved and because of absence of recovery in the second case.

ARNOLD KNAPP.

ARACHNOIDITIS AND VASCULAR COMPRESSION OF THE CHIASM IN A TABETIC PATIENT. M. DAVID, E. HARTMANN and E. HÉBERT, Bull. Soc. d'opht. de Paris, December 1936, p. 789.

The authors report the case of a man aged 48 who was known to be tabetic for four or five years. Gradually there was loss of vision in the lower quadrants of the fields, associated with inability to recognize objects below the usual level of the eyes. Vision was reduced to 2/10 for the right eye and 3/10 for the left eye. General antisyphilitic treatment failed to increase the vision or the fields, but apparently there was no increase in the atrophy of the optic nerve. Exposure of the chiasm revealed optochiasmic arachnoiditis. The anterior cerebral artery was fixed by fibrous strands to the membranes at the crossing. The compression was relieved, and the fields increased markedly in size. Visual acuity was enhanced from 2/10 to 3/10 for the right eye and from 3/10 to 5/10 for the left eye. This is the first reported case in which syphilitic meningitis surrounding the chiasm in a tabetic patient has been treated by operative means. A photograph of the operative field and charts of the visual fields are included.

L. L. MAYER.

Operations

A New Operation for Chronic Glaucoma. O. Barkan, Am. J. Ophth. **19**: 951 (Nov.) 1936.

Barkan gives a brief history and critical survey of operations for glaucoma. He refers to his investigations of the cause of glaucoma and states that in over half of the cases of noncongestive primary glaucoma obstruction of the outflow of aqueous from the anterior chamber into Schlemm's canal causes the condition. This obstruction is located in the sclerocorneal trabeculum. He states that these conclusions are confirmed by incising the trabeculum and opening Schlemm's canal, thus restoring the normal tension. This is the basis of his operation, which consists of opening Schlemm's canal by means of a knife introduced at the temporal limbus and carried across the anterior chamber until its blade disappears behind the nasal limbus. He now uses a specially constructed contact glass and a head loupe, which enables him to follow the knife while it pierces the trabeculum and opens Schlemm's canal. He reports eleven cases in which this procedure was used and gives his conclusions and the following summary:

"It is suggested that this operation, which restores the physiological function of Schlemm's canal, solves the surgical problem of most cases of chronic primary glaucoma. It is equally successful in certain cases of secondary glaucoma. The results are predictable and appear to be permanent. It involves a new principle in the surgery of glaucoma in that the angle of the anterior chamber and Schlemm's canal are under full view and magnified during the operation. The operation is without danger when the proper technique is used and has proved completely successful in the writer's hands when certain preoperative indications (binocular biomicroscopic diagnosis) have been fulfilled."

W. S. Reese.

Keratoplasty from Cadaver's Cornea. M. I. Averbach, Vestnik oftal. **10**: 345, 1937.

Averbach discusses the advantages of using the cadaver's cornea for corneal transplantation. He recommends the use of the cadaver's conjunctiva (Filatov's method) for a conjunctival flap instead of the host's conjunctiva; by this procedure a large defect in the conjunctiva is avoided, and less trauma is produced in the host's eye. The cadaver's conjunctiva should be saved from injury as much as possible.

O. Sitchevska.

Orbit, Eyeball and Accessory Sinuses

The Optic Foramen. J. Gray Clegg, Brit. J. Ophth. **20**: 667 (Dec.) 1936.

This article has to do largely with the roentgenology of the optic foramen. Clegg reviews the literature and reports a case of his own, that of a man aged 25. Visual acuity was 6/6 and blurred in each eye. There was a large absolute central scotoma. The foramen

measured 3 mm. in the right eye and 4 mm. in the left. The optic disks were extremely pale.

The author concludes: "Careful and accurate radiography should be carried out in all cases of affection of the optic nerves. Positive evidence of variations in the lumen and in the state of the walls of the optic foramina is of considerable help, but negative findings are also of much service in diagnosis and treatment."

W. Zentmayer.

Ophthalmologic Sequelae in the Radical Cure of Fronto-Ethmoidal Sinusitis. P. Halbron, Bull. Soc. d'opht. de Paris, October 1936, p. 636.

The ocular complications following operation on the frontal sinus are well known. Halbron reviews such complications in forty-eight patients. These are grouped into four classes: (1) difficulty with motility of the globe, (2) lacrimal complications, (3) palpebral complications and (4) corneoconjunctival complications.

In four of Halbron's patients the complications fall in the first group. Diplopia was the leading symptom. He cautions that treatment must be delayed, as restitution may occur even as late as three months following the operation. Graphs of the muscular balance in these four patients are shown. Interference with lacrimation is most likely due to subcutaneous inflammation. Complications affecting the lid are frequent. Even after healing, inflammation may persist at the internal angle of the lid. Infection of the conjunctiva has been reported, but this did not occur in Halbron's cases. Keratitis without ulceration, however, was frequent.

Halbron concludes that complications may be minimized if the rhinologist keeps in mind the following points:

1. The proper incision avoids the lacrimal sac by passing under and to the side of it.

2. The periosteum of the orbit is removed except in the region of the pulley of the superior oblique muscle.

3. The lids must be held in proximity following the operation.

A bibliography is appended.

L. L. Mayer.

Diagnosis of Fractures of the Sphenoidal Wings. K. Velhagen, Arch. f. Augenh. 110: 365, 1937.

Velhagen examined a series of skulls at the Anatomical Institute at Halle and classified the sutures formed by the sphenoid bone as zygomatic and frontal. These were arranged in three different types. The difficulties of diagnosing fractures in this region are mentioned.

F. H. Adler.

Ectogenous Infection of the Eyeball with Bacillus Phlegmonis Emphysematosae (Welch-Fränkel). H. Rieger, Arch. f. Ophth. 137: 61 (April) 1937.

Rieger reports five cases of septic endophthalmitis following perforating injury. The condition was characterized by a fulminant course and

by general malaise with fever. By culture of material obtained by puncture of the vitreous Bacillus Welchii was demonstrated to be the cause of the endophthalmitis. In such infections the fulminant course often makes evisceration of the globe necessary.

P. C. Kronfeld.

Physiology

An Investigation of the Angular Relation of the Visual (Visierlinie) and Optic (Corneal) Axes of the Eye. G. E. Park, Am. J. Ophth. 19: 967 (Nov.) 1936.

This technical article does not lend itself to abstracting. The following conclusions are stated:

"1. The described method of securing coincidence of the optic axis of the telescope with the visual axis is accurate to within a maximum error of one-fourth degree.

"2. The amount of error in identifying the visual axis by means of the red spot is of the same order as given above.

"3. The angular relation of the optic and visual axes is a variable which accounts for different readings over a series of observations or consecutive readings within a short period.

"4. These variable readings can scarcely be attributed to any corneal change nor to a change in the anatomical position of the fovea.

"5. This leaves but one factor within the eye to account for such variation, the crystalline lens. Possible unequal action of the ciliary muscle, causing prismatic action of the lens by excessive compression at a portion of the lens margin or bodily decentration of the lens from the then existing visual axis, would account for such variation.

"6. The described effect of eserine is entirely upon the ciliary muscle, which seems to indicate this variation to be a result of unequal action of the ciliary muscle.

"7. There appears to be a definite coordination between the ciliary and extrinsic muscles in establishing and maintaining fixation."

W. S. Reese.

Caliber of the Retinal Vessels in Health and Disease. E. Lobeck, Arch. f. Ophth. 136: 439 (Feb.) 1937.

Lobeck's method for the determination of the caliber of the retinal vessels (*Arch. f. Ophth.* 133: 152, 1934) is based on the principle of heliometry. Double images of the fundus are produced by cutting the eyepiece of Gullstrand's ophthalmoscope into halves and by letting the half lenses slide against one another in the plane perpendicular to the optical axis. This displacement is done and measured by a micrometer screw. To estimate the width of a part of the fundus one of the images is displaced until the displacement equals the width to be measured. The micrometer readings are transposed into millimeters by substituting 1.5 mm. for the diameter of the disk, which is measured in micrometer scale units in every case. With this method the width of the

first branches of the central artery and vein were measured for a number of normal persons and for patients with various kidney diseases. The latter diseases were classified according to Volhard's classification. Patients with cardiac decompensation were excluded. The results are given in the table.

Caliber of the Retinal Vessels in Normal Persons and in Patients with Kidney Disease

Diagnosis	Width of the Retinal Vessels in Mm.		Relation in Caliber Between Arteries and Veins
	Arteries	Veins	
Normal condition	0.134-0.088	0.176-0.097	1:1 to 1:1.4
Red hypertension	0.127-0.074	0.140-0.09	1:0.9 to 1:1.45
Pale hypertension	0.081-0.053	0.138-0.097	1:1.5 to 1:2
Nephrosclerosis	0.058-0.027	0.131-0.092	1:1.57 to 1:4
Nephrosclerosis with albuminuric retinitis	0.033-0.026	0.095-0.091	
Acute nephritis	0.133-0.071	0.161-0.09	
Chronic nephritis	0.062-0.053	0.112-0.098	1:1.71 to 1:1.86
Chronic nephritis with albuminuric retinitis	0.084-0.057	0.112-0.100	

Lobeck states: "In all cases of albuminuric retinitis we found a disproportion between the caliber of the retinal arteries and veins. This was more pronounced in the cases of nephrosclerosis than in those of chronic nephritis . . . In diabetic retinitis the retinal arteries had a normal caliber." Acetylcholine, choline and aminophylline produced a measurable degree of dilatation of the retinal arteries, whereas amyl nitrite and perhaps also caffeine dilated the retinal veins.

P. C. KRONFELD.

DIRECTED PERMEABILITY OF THE ELASTIC MEMBRANES OF THE EYE. B. NAKAMURA, Y. UCHIDA and O. NAKAMURA, Arch. f. Ophth. 136:471 (Feb.) 1937.

In experiments in vitro the diffusion of cevitamic acid through the capsule of the lens was quicker in the direction into the lens than in that out of the lens.

P. C. KRONFELD.

Refraction and Accommodation

SUDDEN SPASM OF ACCOMMODATION (INTOXICATION BY METHYLENE BLUE?). J. PLICQUE, Bull. Soc. d'opht. de Paris, October 1936. p. 617.

A patient aged 37 years consulted Plicque because of sudden blurring of his distance vision. The visual acuity of the right eye declined from 9/10 to 3/50, and that of the left eye from 9/10 to 5/50. The pupillary diameter and reflexes were normal. The ocular tension measured 28 mm. No treatment was instituted for twenty-four hours in order to note any change and for the purpose of diagnosis. After that period vision remained the same; the tension receded slightly. Atropine

was instilled, and refraction carried out with the eyes under the influence of this cycloplegic showed the need for a — 1.75 D. sph. for the right eye and a —3.5 D. sph. for the left, but forty-eight hours later the correction for the right eye was + 0.5 D. and the left eye was emmetropic. The urine and the Wassermann reaction were normal. The question of diabetes and psychophysiologic change was investigated, without positive results. Cases of a similar condition that occurred after an operation for strabismus, reported by Plantenga and by Paton, in a patient with parkinsonism, reported by Guillermin and Chams, and in association with facial spasm, reported by Terrien, Schaeffer and Blum, gave no insight into the cause. The patient had been treated with methylene blue (methylthionine chloride) for gonorrheal urethritis. Plicque questions whether the drug's influence on the neurovegetative system, with predominance of stimulation of the parasympathetic system, may not have caused the condition. A bibliography is given.

<div style="text-align: right">L. L. MAYER.</div>

Retina and Optic Nerve

THE LAURENCE-MOON-BIEDL SYNDROME—RECORD OF A CASE. JOSEPH R. MUTCH, Brit. J. Ophth. 21: 225 (May) 1937.

The patient's mother, one of a family of twelve, is living and healthy; two males are affected with atrophy of the retina and optic nerve. The patient is the third of five children, four being males and one a female. There was no parental consanguinity. The remaining two brothers and the sister are alive and normal.

The patient is aged 35. His vision became dim at the age of 12 years but later improved, only to fail again at the age of 30. Night blindness is not present. Vision is 6/60 in each eye. The fields are contracted and show a defect in the left upper temporal area. There is an indefinite central scotoma for red and green. He is not color blind. Each optic disk is pale and has sharp edges. There are a slight mottling at both maculae and a gray spot near the left macula but no pigmentary changes at the periphery. The other symptoms are: moderate obesity, hypogenitalism and hypospadias, mental retardation, polydactyly and syndactyly. The article is illustrated.

<div style="text-align: right">W. ZENTMAYER.</div>

SYNDROME OF DIRECT COMPRESSION OF THE INTRACRANIAL PORTION OF THE OPTIC NERVE. PIERRE DESVIGNES, Ann. d'ocul. 174: 289 (May) 1937.

As indicated by the title, this article deals with cases in which a tumor, either fibrous or vascular, presses on the part of the optic nerve just in front of the chiasma. The optic nerve is fixed here by its continuity with the chiasma and by the optic canal and the arteries of this region, the ophthalmic artery outside and below and the anterior cerebral artery and the anterior communicating artery above. In such a position the optic nerve cannot escape from pressure, as it might do in the orbit, where it is surrounded by soft tissue. It therefore follows, if a tumor begins in this neighborhood, that the nerve will soon be compressed, and ocular signs will first draw attention to the condition.

The signs of compression of the chiasma are well known, but those of compression of the optic nerve have not been as much studied. In some cases study has established the diagnosis of a cerebral tumor localized near the optic nerve, and in such cases if operation has not been too long delayed patients have escaped rapid blindness and death accompanied with great suffering.

The author gives first a historical review of the subject, followed by a discussion of the first clinical signs and of (1) vision, (2) the visual fields, (3) the appearance of the fundus and (4) other ocular symptoms. There is a bibliography.

<div align="right">S. H. McKee.</div>

A CASE OF DETACHMENT OF THE RETINA WITH A HOLE IN THE MACULA: TREATMENT BY DIATHERMY. L. MAMOLI, Ann. d'ocul. 174: 309 (May) 1937.

This article consists of a short report of the case of B. J., 31 years old, of delicate health, who was found to have the retina detached completely in its inferior part. There was bilateral myopia of 13 D., with bilateral macular chorioretinitis. There was present a macular hole across which could be seen the displaced choroid. Because of the much reduced vision, the age of the patient and the presence of the hole, operation was decided on, but the various known technics did not give satisfactory results.

The patient was treated by diathermy coagulation of the macular region, obtained by the use of an electric needle introduced into the interior of the globe and pushed by ophthalmoscopic control to the retinal region.

<div align="right">S. H. McKee.</div>

THE PROBLEM OF PAPILLARY EDEMA OF HYPERTENSIVE PATIENTS. A. COUADAU, Bull. Soc. d'opht. de Paris, July 1936, p. 579.

Edema of the disk is the primordial and primitive sign of the papilloretinitis of hypertensive patients. Associated with hemorrhages and exudates of the retina, it completes a typical ensemble which is easily recognized and has a grave prognosis. Couadau has especially studied isolated edema of the papilla. According to its intensity it assumes two forms—simple edema and edema due to stasis. Simple edema is frequently observed, seventeen cases having been under the author's observation in a few years. Edema of the disk due to stasis necessitates differentiating between that due to papillitis of hypertensive patients and that due to a cerebral tumor associated with intracranial hypertension. Two cases are reported in which signs of nephritis were not present in the early course but appeared later. In cases of true tumor the edema of the disk is progressive. No signs of nephritis are ever present. The retinal arterial tension is raised in papillitis of hypertensive patients but fluctuates in stasis.

Summarizing, Couadau feels that differentiation depends on the findings of general examination and the observations of biochemical study and on the progress or course of the condition. A bibliography is appended.

<div align="right">L. L. Mayer.</div>

Angioid Streaks of the Retina and Pseudoxanthoma Elasticum. A. V. Denti, Ann. di ottal. e clin. ocul. **65**: 93 (Feb.) 1937.

The literature on angioid streaks of the retina associated with pseudoxanthoma elasticum (the syndrome of Groenblad and Strandberg) is reviewed. Denti's patient, a woman of 37, showed a typical form of each condition. Vision was reduced to 1/2. Aside from the typical streaks about the disks, the fundi showed hemorrhages and a number of fine white spots, especially in the central region. Examination of other members of the family gave negative results. During six months numerous fresh hemorrhages were observed in the internal layers of the retina, but none were so deep as the streaks. The author interprets the streaks as abnormal vessels of congenital origin in the deeper retinal layers, perhaps in connection with the choroidal veins. He believes that the degeneration of elastic tissue which is known to cause the cutaneous lesions also causes degeneration in the walls of the retinal vessels and in the lamina vitrea of the choroid, such degeneration being responsible for the hemorrhages and white deposits.

S. R. Gifford.

Significance of Dynamometric Studies for the Explanation of the Mechanism of Choked Disk. J. Sobański, Arch. f. Ophth. **137**: 84 (April) 1937.

In 1934 Sobański published in Polish a monograph on the mechanism of choked disk (reviewed in *Zentralbl. f. d. ges. Ophth.* **31**: 540, 1934). The theory elaborated therein was based chiefly on dynamometric studies, but was also based on experiments on animals in which pressure was exerted on the orbital portions of the optic nerve by means of a clamp. The dynamometric studies demonstrated that the development of a choked disk is conditioned on a disproportion between the pressure in the central vein and that in the central artery. Normally this relation is 1:3. If the intracranial pressure rises, the retinal venous pressure always rises also. If the arterial retinal pressure rises at the same rate no choking develops, despite the increased intracranial pressure. If the arterial pressure does not rise sufficiently so that the ratio between the venous pressure and the arterial pressure becomes 1:1.5, papilledema develops. In this article Sobański restates his views and objects to theories based only on pathologic observations.

P. C. Kronfeld.

Influence of Red Rays on the Function of the Retina. H. M. Dekking, Arch. f. Ophth. **137**: 153 (April) 1937.

The sensitivity for all visible rays of the dark-adapted foveal region of the human retina is lowered by simultaneous incidence of visible rays of a wavelength over 6,200 angstrom units. This phenomenon is most pronounced for minimal intensities of light.

P. C. Kronfeld.

Further Observations on Retrobulbar Neuritis in the Chinese, Chen P'an and W. P. Ling, Chinese M. J. **50**: 1373 (Oct.) 1936.

The authors report the study of seventy patients with retrobulbar neuritis admitted to the wards from a total number of one hundred

and eighty patients with this condition who were seen. In all the cases the pupils were dilated for examination of the fundi. The findings were normal in all cases except five, in which the patients showed retinal hemorrhages without other changes.

The visual fields were examined carefully; there were no abnormal findings. A central scotoma was practically the only sign of retrobulbar neuritis present in all the cases and was usually a relative one for both white and colors; it was confined to the fixation point and was not larger than 10 degrees all around.

The authors conclude that the etiologic factor of retrobulbar neuritis in the Chinese still remains a mystery. They believe that the term "retrobulbar neuritis" applied to this condition may be misleading and think that the phrase "retrobulbar changes of the optic nerve" is preferable. The disease runs a tedious course; the prognosis is usually good, no case of blindness having been met with as yet. There is no special treatment for the condition, and the general condition of the patient is, as a rule, good.

S. H. McKee.

PSEUDO-ATROPHY OF THE OPTIC NERVE IN YOUNG INFANTS. K. T. A. HALBERTSMA, Nederl. tijdschr. v. geneesk. 81: 1230, 1937.

Halbertsma reports the clinical history of a girl 8 weeks old who was brought to him by the parents with the complaint that the eyes had a peculiar staring expression which had made them fear that the child might be blind. The ophthalmologic examination revealed an abnormal behavior of the pupils and a peculiar coloration of the optic papillae. On the basis of another examination made three weeks later, the disorder was diagnosed as pseudo-atrophy of the optic papilla probably resulting from retarded myelinization of the pupillomotor fibers of the optic nerve. Several successive examinations disclosed gradual improvement in that the pupillary reactions became more normal and the peculiar coloration of the papillae began to disappear. The author points out that the condition in this case corresponds to that described as pseudo-atrophy of the optic nerve in the new-born by Beauvieux in 1926. He also cites the signs which, according to Beauvieux, differentiate pseudo-atrophy of the optic nerve from true congenital atrophy of the optic nerve.

J. A. M. A. (W. ZENTMAYER.)

Trachoma

SUBCONJUNCTIVAL AUTOHEMOTHERAPY IN TRACHOMA. E. S. MARTINEZ, Rev. internat. du trachome 14: 19, 1937.

In two cases of trachoma in which from 2 to 4 cc. of the patient's own blood was injected under the fornix the ocular irritation rapidly subsided and the pannus was favorably affected.

J. E. LEBENSOHN.

TRACHOMATOUS KERATITIS. S. SPYRÂTOS, Rev. internat. du trachome 14: 23, 1937.

The statistics of Athens show a definite recrudescence of trachoma and trachomatous keratitis in October, probably owing to intercurrent

catarrhal infections. As the patients grow older the proportion of corneal complications steadily increases.

<div align="right">J. E. LEBENSOHN.</div>

CONSTITUTION IN RELATION TO TRACHOMA. F. MASSOUD, Rev. internat. du trachome 14: 42, 1937.

Very young children acquire trachoma more readily than adults. Lymphatic children suffering from adenoids seem most susceptible. Special sanatoriums for persons with trachoma have been established in Italy, in which the value of sunshine, fresh air and proper food is emphasized.

<div align="right">J. E. LEBENSOHN.</div>

THE CONTAGIOUSNESS OF TRACHOMA CLINICALLY CONSIDERED. J. SÉDAN, Rev. internat. du trachome 14: 65 (April) 1937.

Trachoma is especially contagious in infants, but contagion is possible in persons of all ages, though it occurs seldom in children of school age and only rarely in adults. Important predisposing factors are neglect in personal hygiene, intercurrent conjunctivitis, disease of the lacrimal apparatus and ocular traumatism. Patients with trachoma in boarding schools, barracks and hospital or hotel service should be closely supervised or excluded. The incidence of trachoma is greatest where the general standard of living is lowest.

<div align="right">J. E. LEBENSOHN.</div>

TRACHOMA AND INFECTIONS OF THE LACRIMAL PASSAGES. A. ALIQUÒ-MAZZEI, Ann. di ottal. e clin. ocul. 65: 38 (Jan.) 1937.

Aliquò-Mazzei examined three hundred and eighty patients with trachoma with especial reference to involvement of the lacrimal passages. In only 12 per cent of the cases was there moderate interference with the passage of the fluid into the nose, and in only 5 per cent of cases was there complete obstruction. These were cases of old trachoma and extensive scarring of the lids, with obliteration of the canaliculi or tear points. Dacryocystitis was present in only 4.21 per cent of the cases. In these cases (sixteen) the lacrimal sac was removed. Thirteen patients showed the inflammatory changes commonly seen in dacryocystitis, with the presence of follicles. In three cases true follicles were present in the walls of the sac. There was nothing characteristic about the history. The author concludes that the dacryocystitis did not have any causal relationship with trachoma.

<div align="right">S. R. GIFFORD.</div>

Tumors

CONJUNCTIVOPALPEBRAL TUMOR (DERMOLIPOMA) OF EXCEPTIONAL DEVELOPMENT IN A NEW-BORN INFANT. M. A. DOLLFUS, Bull. Soc. d'opht. de Paris, October 1936, p. 598.

In an otherwise normal new-born infant a pedunculated tumor was noted outside the outer canthus. It was red at birth but on twisting of the pedicle assumed a violet color. It was the size of a large olive, regular, palpable and mobile on the cheek. The conjunctival stalk appeared to have its origin in the cul-de-sac, but a few strands con-

tinued to within a millimeter of the corneal limbus. Pathologic examination confirmed the clinical diagnosis of dermolipoma, as the many and various blood vessels, the sebaceous glands and other dermal elements would testify. A drawing is included. The exceptional size and location are the cause for the report.

L. L. MAYER.

TUMORS OF THE ORBIT: ORBITOFRONTO-ETHMOIDAL OSTEOMA. Q. DI MARZIO, Riv. oto-neuro-oftal. 13: 393 (Sept.-Oct.) 1936.

Di Marzio describes four cases of orbital osteoma located chiefly in the frontal sinuses. In the first case he made a linear incision from the root of the nose along the superior margin of the orbit as far laterally as the tail of the left eyebrow, continuing the incision from the head of the left eyebrow across the glabella to the head of the right eyebrow. The periosteum was separated; the bone was attacked with a hammer and chisel, and excavation was made down to the internal angle of the left frontal sinus, to the root of the nose and along the arch of the orbit, where the tumor was strongly attached. Since the tumor was very large and invaded the frontal sinuses, it was necessary to separate it from its superior wall; the dura was almost completely exposed, pulsating but not perforated.

The tumor had invaded the left as well as the right frontal sinus, extending toward the ethmoid sinuses. The cavities of the sinuses were accurately curetted, and drained by way of the left nostril and the original orbital wound. The deep layers were sutured with catgut, the superficial layers with silk. The tumor, which was found to be an osteoma, measured 5.5 by 5.2 by 4.5 cm. and weighed 68 Gm.

In the second case, since the mass was confined chiefly in the inner angle of the left eye, Di Marzio made a curvilinear incision from the external third of the superior orbital margin to the region of the lacrimal sac, detaching the periosteum down to the ethmoid sinus, where the neoplastic mass was located, and then down to the lacrimal fossa, and separating the lacrimal sac. With a hammer and chisel he then removed the floor of the frontal sinus and the wall of the ethmoid sinus so as to cut out the tumor, which was closely adherent to these structures. He sutured the periosteum and skin, and applied drainage through the nose. This tumor was smaller than that in the first case, measuring 3 cm. in every diameter.

In the third case the tumor was located on the floor of the right frontal sinus and extended down to the ethmoid sinus. The technic of the operation was the same as in the second case.

The fourth case was interesting because six years previously the region over the left eye had become swollen and the patient had fever. At that time he was operated on for orbital abscess, but the exophthalmos did not regress. Four years later the eye again became swollen; the swelling subsided following hot applications.

Roentgen examination at this time revealed a dense opacity in the region of the left orbital margin, which extended to the right orbital margin, invading most of the left orbit and part of the frontal bone through the frontal sinus and reaching up to the anterior cranial fossa. The patient was operated on, with the part under local anesthesia; an

incision was made along the left eyebrow and down to the bone, and the external wall of the frontal sinus was chiseled out. A large lobate mass was seen adhering to the inner wall of the sinus. The rest of the cavity was filled with pus and granulation. The tumor was removed piecemeal with a bone forceps, a very thin long sheet being left, through which the meninges could be seen pulsating. The granulations were removed by curettage, and the sinus cavity was carefully cleaned and drained through the nasofrontal canal and through the exterior wound. This tumor was large, measuring 6 by 4 by 4 cm.

Di Marzio stresses the value of complete removal of the tumor and curettage of the mucosa of the sinuses in order to prevent the inflammation from spreading from the sinuses to the cranial cavity.

He discusses the pathologic features of the neoplasm and concludes, agreeing with Lagrange and Borst, that osteoma of the orbit may have several origins, which must be distinguished in each case

G. Bonaccolto.

Vision

VISUAL TASKS IN SIGHT-SAVING CLASSES. M. Luckiesh and F. K. Moss, Am. J. Ophth. 19: 992 (Nov.) 1936.

This article is based on the determination of actual visual efficiency. Luckiesh and Moss state that the criterion of visual acuity is faulty, whereas measurements of visibility are fundamentally sound. Size of type as a factor in seeing was appraised by measurements of visibility made with the Luckiesh-Moss ophthalmic sensitometer. The authors state that by their technic it is possible to classify pupils in sight-saving classes into a limited number of groups and provide each group with type of the proper size.

W. S. Reese.

Vitreous

THE IMPORTANCE OF DISEASES OF THE CHOROID FOR DEVELOPMENT OF DETACHMENT OF THE VITREOUS. H. Rieger, Arch. f. Ophth. 136: 119 (Nov.) 1936.

To the long series of articles dealing with detachment of the vitreous published by pupils of Lindner, Reiger adds a clinical study of 356 persons suffering from diseases of the fundus in which detachment of the vitreous is known to be common. In addition to a thorough ophthalmoscopic examination of the fundus, the vitreous was examined ophthalmoscopically and with the slit lamp, with and without a contact glass. The data thus obtained indicate that detachment of the vitreous is common in, and probably caused by, diseases of the choroid. A large number of interesting and new slit lamp pictures of detached vitreous are described.

P. C. Kronfeld.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION OF OPHTHALMOLOGY

April 15, 1937

CHARLES R. HEED, M.D., *Chairman*

ALEXANDER G. FEWELL, M.D., *Clerk*

ANESTHESIA INDUCED WITH TRIBROMETHANOL IN OCULAR SURGERY. DR. EDWARD W. BEACH.

Tribromethanol when used to induce anesthesia in surgical intervention on the eye reduces the psychic shock because it allows administration in the patient's room and produces a quiet sleep. It relaxes the extra-ocular muscles and reduces the intra-ocular tension. The slight fall in the blood pressure which it causes minimizes bleeding during operation and gives a paler conjunctiva than other anesthetics. When employed as a basal anesthetic it requires much less of the supplementary agent than other anesthetics. The recovery of consciousness is prolonged, and this period of amnesia is welcomed by patients with ocular conditions. Because of slow recovery there is need for a longer period of nursing to prevent unconscious movements. There are little or no postoperative nausea and vomiting.

DISCUSSION

DR. CHARLES W. LE FEVER: I have used much larger doses of tribromethanol than those just mentioned by Dr. Beach. I started using smaller doses, averaging from 75 to 80 mg. per kilogram of body weight, but soon found that it was necessary to give the patient ether to complete the anesthesia. This small dose is sometimes sufficient for senile patients. In patients under 40 years of age, of the muscular type, I give 95 mg. per kilogram, and even then it is often necessary to give supplementary ether.

I have never seen any especially unfavorable effects from tribromethanol. In older persons I believe it is the rule that the blood pressure falls rapidly during the first twenty minutes, while the patient is becoming narcotized. At the end of that time, or perhaps within thirty minutes from the time the tribromethanol was given, the drop in the blood pressure stops. Thereafter the blood pressure starts to rise, continuing to do so gradually from this time until the completion of the operation or until it reaches normal. I have seen it fall as low as 56 systolic and 20 diastolic, at which point it responded promptly to 5 minims of epinephrine hydrochloride given by hypodermic injection.

DR. LUTHER C. PETER: I have been using tribromethanol for a number of years and find it one of the best forms of anesthetic for

surgical intervention on the eye.   In fact, it has few limitations, as Dr. Beach has pointed out.   It is not satisfactory for a child under 10 years of age, because of inability of the patient to cooperate.   The upper age limits have not been fixed thus far.   I agree with Dr. Beach, however, that elderly persons and those who are obese should receive the smaller dose of 80 mg. rather than the upper limit of 90 mg.

In the matter of advantages and disadvantages, Dr. Beach has placed lowering of the blood pressure and lowering of the intra-ocular pressure as disadvantages.   On the contrary, I regard these effects as advantages in cases of cataract and glaucoma.   Among the disadvantages, nausea is the one which must receive careful consideration.   In my experience in the Graduate Hospital of the University of Pennsylvania, there have been few cases of nausea, and in most of these nausea was due to a necessarily long period of anesthesia, as in operations for retinal detachment, when supplementary anesthesia must be used.   Ether is not a satisfactory supplementary anesthetic.   Divinyl ether and chloroform are the best agents of this kind.

The great advantage of using tribromethanol in operations on elderly persons, especially those who have cataract, is the fact that it eliminates worry.   The anesthetic is administered in the patient's room.   A second point of great advantage, especially in the more difficult operations, is the quietness of the patient on the operating table.   In fact, tribromethanol has made surgical intervention a real joy, even in the most difficult cases.   I refer to cases of complicated cataract following uveitis, in which there are marked posterior synechiae.   One can make the section without difficulty, release the adhesions with a spatula and deliver the lens in its capsule without any difficulty.   A bridle suture is introduced under the superior rectus muscle to control the eye.

The other type of case in which tribromethanol is advantageous is that of the patient who has had a sclerocorneal trephining performed and who also requires extraction of cataract.   As is known, the anterior chamber, as a rule, is shallow.   But with a narrow cataract blade the section can be made and a conjunctival flap fashioned; the wound can be enlarged by straight scissors; synechiae, if present, can be released, and the lens can be extracted in its capsule.   In fact, when the patient is perfectly relaxed and the eye is quiet one can do almost any type of difficult surgical intervention.

Choroideremia: Report of a Case.   Dr. Walter Lillie.

Arthur J. Bedell in his article on choroideremia (Arch. Ophth. **17:** 444 [March] 1937) stated:

"The subject of this essay is a peculiar condition characterized by night blindness, sometimes evident in early life, but often not conspicuous until the third or fourth decade.   It seems to be familial, affecting the males.   .   .   .   Choroideremia is an entity with a pathognomonic fundus, and as it develops during the life of the patient it must not be considered as a congenital absence of choroid but as a dissolution of that membrane.   .   .   .   A large portion of the choroid is absent; the macula is intact, and the vessels of the choroid are small and straight or

entirely absent. There is no ectasia of the sclera. . . . . The macular region is the last to register the destructive process."

Report of Case.—A man aged 70 years was referred to me by Dr. F. W. Uhler, of Easton, Pa., because of diplopia for the past three months. The patient had always been in good health. For twenty years he had worn bifocal lenses with satisfaction. During the past three years he had been able to carry on as road supervisor without difficulty, although he was conscious of scintillating scotoma in the form of "heat waves," and when the bright sunlight was reflected from buildings "red vision" resulted. His glasses were satisfactory, although ocular fatigue ensued after half an hour of reading. One year previously, during a period of three months he lost 20 pounds (9.1 Kg.), but he had been well since October 1936. My examination, on Feb. 23, 1937, revealed vision to be 6/10 in the right eye and 6/12 in the left eye, with his glasses. The correction for the right eye was a + 0.12 D. sph. combined with a + 1.00 D. cyl., ax. 92, and that for the left eye was a + 0.25 D. sph. combined with a + 0.75 cyl., ax. 90, with an additional + 2.50 D. for near work on each lens. The pupils were equal and the reflexes normal. The ocular movements revealed slight weakness of the left internal rectus muscle and loss of convergence. The ophthalmoscopic examination revealed a few small opacities in the vitreous in each eye. The optic disks were normal in color, and the margins were distinct, but there was loss of the choroid in the peripapillary area. The retinal arterioles were slightly attenuated. The choroid was absent in the lower portion of the fundus in each eye and in an area extending into the superior nasal portion in the right eye. The border was distinct, and the retinal arteries extended uninterrupted over this area. Clumps of pigment were fairly numerous but well scattered. A few choroidal vessels still remained, which disappeared abruptly into the normal choroidal margin. The sclera appeared normal. The macular region in each eye was intact. Tests of the visual fields revealed definite bilateral superior altitudinal anopsia. The color vision was normal as tested with the Ishihara charts. The general examination revealed nothing of importance, and the patient's health was excellent for this age group. There had apparently been no similar familial visual disturbance in the past two generations. This case is of interest, as it not only furnishes an additional instance in the small group of cases of choroideremia already published but apparently is the case in which the visual disturbance had the oldest onset, with no night blindness, "red vision" from reflected sunlight being a disturbing feature. The diplopia was probably a coincidental condition of the central vessels of the retina.

PARTIAL BILATERAL COLOBOMA OF THE OPTIC NERVE. DR. FRANCIS HEED ADLER.

Complete coloboma of the optic nerve seldom offers any difficulties in diagnosis. The porus opticus is at least two or three times the diameter of the normal disk, and the cup is often 3 or 4 D. in depth. Associated with anomaly of the disk there is frequently a coloboma of the choroid. In cases of partial coloboma of the optic nerve, however, there may be

Our investigations led us to believe that the causes responsible for secondary glaucoma give a strong hint as to the pathologic physiologic condition incident to primary glaucoma.

Dr. Gross' survey points out the ineffectiveness of the present methods of treating atrophy of the optic nerve that is the result of syphilitic infection. His report indicates that the ophthalmologist must look in other directions for newer methods in collaboration with the syphilographer.

Both these investigations should give encouragement to the younger ophthalmologists to carry out research based on clinical records coupled with examination of the patients whom these records concern.

EFFECT OF DISTANCE USED IN TESTING ON THE APPARENT SIZE OF A SCOTOMA. HAZEL WENTWORTH, PH.D.

The effect of the distance used in testing on the apparent size of a scotoma, as indicated by changes in the size of 100 normal blindspots for form and color when outlined at different distances from the eye, was described.

The exact area of the blindspot in 100 different eyes was obtained for 1 degree Hering white, Heidelberg blue, red and green test objects at distances from the eye of 16.6, 33 and 100 cm., respectively, and exactly measured in an effort to determine whether the distance from the eye at which a scotoma is mapped has any effect on its apparent size, that is, on the visual angle subtended, and. if so, to what extent, and whether there is any differential color effect.

The work was done on the Holloway-Cowan screen under carefully controlled conditions as to the illumination, the brightness of the background, the method of mapping, the size of the test object, the amount of practice and other factors.

With the blindspot obtained at 33 cm. taken as the standard size for comparison, it was found that the size of the average blindspot increased relatively at the distance of 16.5 cm. and decreased relatively at the distance of 100 cm. The total decrease in size for the average blindspot at a distance between 16.6 and 100 cm. from the eye was 3.5, 8, 22 and 27 per cent for form, blue, red and green, respectively, as compared to a mean error in size for the same number of repeated tests at the same distance (33 cm.) of 2.5, 2.8, 3.3 and 3.4 per cent, respectively.

From tables showing the distribution it was found that for red and green 8 or 9 of every 10 subjects showed an average decrease in size of nearly one third that taken as the standard when the distance was increased from 16.6 to 100 cm. from the eye; for blue, 2 of 3 subjects showed an average decrease of one fourth the standard area, and for form slightly over one half showed an equal amount of change. Only about four fifths as many patients showed a decrease in size when the distance was increased from 16.6 to 33 cm. from the eye as when it was changed from 33 to 100 cm. The changes in apparent size with changes in distance were considered, on theoretical grounds, to be chiefly the result of changes in accommodation. They were shown to

be valid from the statistical standpoint. The causal factor of the differential color effect could not be definitely determined.

The significance for practical perimetry was pointed out, i. e., the fact that not only does the area of a scotoma obtained at different distances from the eye represent different retinal areas when a constant visual angle is assumed but also, since the change is differential for form and color and between the colors, the relative areas of partial and complete loss of sensitivity vary at the different distances. The factor of distance was shown to be of particular importance in the use of a red test object, since red is the color showing the greatest change with distance and a test with this object is also the most reliable sensitive test of lesions in the optic nerve fibers and tract which are not observable on objective examination. The importance of the differential effect is shown in that, when attempting to determine the extent to which the nerve fibers have become involved in a retinochoroidal lesion, as indicated by the relation of the limits of the scotoma for red and for blue, respectively, the area of the scotoma for red will be judged relatively smaller as compared to that for blue at the greater distance. A plea was therefore made that for comparable results a standard distance be adopted.

This paper, presented in tribute to the late Dr. T. B. Holloway, who suggested the problem and whose support made it possible to carry out the work, will be published in full at a later date.

DISCUSSION

DR. FRANCIS HEED ADLER: Just what causes the change that Dr. Wentworth has outlined no one can say. Whether accommodation is an important factor could perhaps be determined by examining a number of patients under the influence of a cycloplegic.

DR. SIDNEY L. OLSHO: Before scientific conclusions can be drawn I should like to ask if it would not be necessary to insure that the lighting be so controlled that the test objects receive the same amount of illumination at the two unequal ranges, that of the campimeter and that of the tangent screen.

DR. HAZEL WENTWORTH: The illumination of the screen was kept constant at 7 foot-candles at all distances. The Holloway-Cowan screen was designed to make this possible.

DR. WALTER I. LILLIE: If the size of the blindspot decreases at increased distances it might be of interest to know whether the peripheral field varies at similar distances.

DR. HAZEL WENTWORTH: No similar studies have been made of the peripheral field. I have found it difficult at times, however, to decide whether or not a certain increase in the amount of contraction of a field as the distance increased was definitely tubular.

DR. ALFRED COWAN: I should like to know whether changes in the pupil were taken into consideration, as these might possibly be the factor.

DR. HAZEL WENTWORTH: Changes in the pupil were considered as a part of the general accommodative effect.

errors in diagnosis.   A case of partial coloboma of each optic nerve
was reported, in which the condition had been diagnosed as primary
optic atrophy and the patient had been referred to Dr. Francis Grant
as possibly having a pituitary tumor.

COLOBOMA OF THE CHOROID AND PARTIAL COLOBOMA OF THE
    DISK. DR. ALEXANDER G. FEWELL and DR. EDWARD S.
    GIFFORD JR.

J. K., aged 8, presented in the right eye a congenital crescent on
the nasal side of the disk and a hole in the disk on the nasal side.   Below
the disk was a large coloboma of the choroid, oval and located at 180
degrees, separated by a narrow strip of normal fundus from another
choroidal coloboma below which it extended into the periphery as far
as could be seen.   In the left eye there was a small, round coloboma
of the choroid, extending down and out from the disk.

M. K., aged 16, a sister of this patient, showed a hole in the disk
on the temporal side in the left eye.

The fundi of each parent were normal, the mother being hyperopic
and the father myopic.   Of 6 living children, record of only 4 could be
obtained.   The 2 just cited as presenting anomalies were hyperopic; 2
others, who had normal fundi, were myopic.

REVIEW OF GLAUCOMA AT THE WILLS HOSPITAL, PHILADELPHIA.   DR.
    LOUIS LEHRFELD and DR. JACOB REBER.

This article will appear in a later issue of the ARCHIVES.

A STATISTICAL INVESTIGATION OF SYPHILITIC ATROPHY OF THE
    OPTIC NERVE.   DR. LOUIS LEHRFELD and DR. ELMER R. GROSS.

A ten year survey of syphilitic atrophy of the optic nerve at the
Wills Hospital included study of 552 patients with primary atrophy of
the optic nerve and 48 with secondary atrophy of the optic nerve.   Of
these 600 cases, 91 were studied and observed sufficiently for con-
clusive evidence to be obtained.   The remaining cases were tabulated
for statistical purposes.   The purpose of this survey was to determine
what happened to these patients with syphilitic involvement of the optic
nerve from a therapeutic, prognostic and social-economic standpoint.

The patients were divided into three main groups.   Group I
consisted of those patients who had received no treatment for the
atrophy of the optic nerve.   The patients in this group were blind
or nearly blind on admission to the clinic.   Group II was composed of
those patients who had received antisyphilitic treatment.   Group III
was made up of those patients who had received a special form of
treatment, including fever and subdural therapy.

Seventy-four and nine-tenths per cent of the patients in group I
were blind in less than three years, and all were blind at the end of
a five year period.   Twenty-three and eight-tenths per cent of the
patients in group II were blind in less than three years, and all were
blind at the end of eight years.   Twenty-eight per cent of the patients
in group III were blind in less than three years, and all were blind at

the end of eight years. This refers to the observed cases from the onset of the symptoms to blindness. It appears that primary atrophy of the optic nerve is a progressive process, despite treatment. The present day therapeutic armamentarium does not offer a great deal with which to combat this grave complication of syphilis.

A general statement cannot be made that treatment is contraindicated in primary atrophy of the optic nerve; although not 1 patient was considered improved in our series, the condition in 8 remained stationary with routine treatment and that in 11 remained stationary with subdural treatment. It is, however, likely that these patients may be blind at the end of a ten year period.

The result of the subdural treatment depends primarily on the irritant effect of the substances introduced intraspinally. A modification of the Swift-Ellis treatment that we employ in the clinic at the Wills Hospital seemed to produce more meningeal irritation, which manifested itself in signs and symptoms of aseptic meningitis. The patients who have had these reactions seem to hold their own better than those who have not.

The onset of atrophy of the optic nerve in most instances is so insidious that the person does not realize the seriousness of the situation. When the patient complains of blurring of vision, the atrophy of the optic nerve is usually already pronounced. The early concomitant signs and symptoms of the associated neurosyphilis are so mild that they frequently escape the physician's attention.

The present day treatment of syphilitic primary atrophy of the optic nerve is unsatisfactory. As in all forms of syphilis, prophylaxis is the best form of treatment. This can be accomplished by education of the public and the physician. Measures to prevent atrophy of the optic nerve should include, first, early diagnosis and adequate treatment of early syphilis and, second, early diagnosis and adequate treatment of neurosyphilis. Pupillary abnormalities are often the first objective signs of neurosyphilis, and any patient in whom these are observed should be subjected to a thorough examination, including a test of the spinal fluid. The diagnosis and treatment of atrophy of the optic nerve require close cooperation between the syphilologist and the ophthalmologist. Plotting of the visual fields, examination of the fundus and tests of visual acuity are of prime importance in diagnosis and in determining the severity of the atrophy of the optic nerve.

DISCUSSION

DR. LOUIS LEHRFELD: The reports presented by Dr. Gross and Dr. Reber demonstrate that all research in ophthalmology is not confined to the laboratory. At the Wills Hospital there are many volumes of records which are waiting for investigators such as Dr. Gross and Dr. Reber to delve into their stored-up knowledge and to abstract from them information which may be just as important to ophthalmology as researches made in the laboratory.

Dr. Reber's report states that in 22 per cent of the patients with glaucoma who reported at the Wills Hospital in the last ten years the condition was secondary. Generally, one dismisses secondary glaucoma as playing no part in the consideration of the subject of glaucoma.

Our investigations led us to believe that the causes responsible for secondary glaucoma give a strong hint as to the pathologic physiologic condition incident to primary glaucoma.

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Dr. Hazel Wentworth: The illumination of the screen was kept constant at 7 foot-candles at all distances. The Holloway-Cowan screen was designed to make this possible.

Dr. Walter I. Lillie: If the size of the blindspot decreases at increased distances it might be of interest to know whether the peripheral field varies at similar distances.

Dr. Hazel Wentworth: No similar studies have been made of the peripheral field. I have found it difficult at times, however, to decide whether or not a certain increase in the amount of contraction of a field as the distance increased was definitely tubular.

Dr. Alfred Cowan: I should like to know whether changes in the pupil were taken into consideration, as these might possibly be the factor.

Dr. Hazel Wentworth: Changes in the pupil were considered as a part of the general accommodative effect.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

London, England, June 11, 1937

Mr. W. H. McMullen, O.B.E., F.R.C.S., *President*

The Epithelial Growths of the Conjunctiva and Cornea. Mr. E. F. King.

There seems to be no essential difference in the pathologic nature of the epithelial growths found on the conjunctiva and on the cornea. The tumors may be either benign or malignant. The usual site for malignant neoplasms is the limbus. The more innocent tumors may assume malignant properties.

Of the innocent tumors, the first is papilloma, which is seen mostly in the fornices and on the caruncle. Each papilla consists of a delicate stroma containing thin-walled vessels, covered by stratified epithelium. This type of tumor shows a tendency to local recurrence after it has been removed, thus resembling similar tumors in the urinary tract, and it may become malignant. Another form of innocent tumor is the epithelial plaque, which occurs on the conjunctiva or the cornea as a well defined area of epithelial hyperplasia, superficially keratinized. Parsons considers the plaques congenital and dermoid in nature. Lister and Hancock regard them as analogous to callosities on the surface of the skin, representing hyperplasia of the horny layers.

Of the malignant growths, rodent ulcer never involves the eye primarily, but only secondarily by extension. The common site for epithelioma is the limbus, but a number of cases have been reported in which it arose wholly on the cornea. Epithelioma on the conjunctiva, at a distance from the limbus, seems to be the result of malignant change in a papilloma. This variety of tumor is most frequently seen in the interpalpebral space, especially on the temporal side, that most exposed to trauma. In several cases this tumor has followed penetrating wounds. The growth is sometimes pigmented by the normal pigment cells at the limbus and at first resembles a limbic dermoid, but at a later stage it ulcerates and is liable to bleed on slight provocation. Bowman's membrane offers considerable resistance to invasion by tumors, but yields later, and then infiltration occurs along the lymph spaces around the corneal lamella. The sclera forms a dense barrier and becomes eroded rather than infiltrated. On reaching Tenon's capsule the growth extends rapidly, and in some cases it completely surrounds the globe. In a high proportion of cases in which the growth is advanced there is intra-ocular extension along the anterior perforating vessels and Schlemm's canal, or by way of the vorticose vessels in cases in which Tenon's capsule in involved. There seem to have been only a few authentic reports of generalized metastasis.

Microscopically, the growth consists of stratified epithelium and stroma in papillomatous formation. The following features are characteristic of malignancy: (*a*) There is variation in the size and chromatin content of the epithelial cells, in some of which the nucleus is fragmented and degenerate, active mitosis being seen in others. (*b*) Penetration of epithelial cells into the underlying tissues is unusual, a

more usual appearance being actual erosion and loss of substance of the cornea and sclera, without infiltration. Some degree of dipping of the epithelium at the normal limbus is normal. (*c*) Characteristic nests of cells are present, which are seen in the fibrous stroma of the growth rather than in the underlying structures. (*d*) There is inflammatory cellular infiltration in and around the mass, even when there is no secondary infection due to ulceration.

With regard to the treatment of carcinoma, when the cornea is extensively involved or the growth has penetrated the globe, excision or exenteration is clearly indicated. If the growth is an early small limbic tumor it is generally agreed that conservatism is permissible, though many urge excision in all cases if the other eye is sound. As to conservative lines of treatment, local excision, cauterization and roentgen therapy (alone or in combination) have resulted in variable success. Radium therapy seems to offer an efficient and simple treatment which is completely successful in most cases and appears to be particularly indicated for this condition, of which an early diagnosis is the rule. Several authorities have published highly satisfactory results of treatment of epibulbar growths by radium. The method of treatment has been, in general, the application of unscreened beta rays to the surface of the growth for short periods, and this is repeated at intervals, if necessary. If the growth is considerable, it is wise to remove first the greater part of it surgically.

Radiation cataract has been reported by many observers after radium has been used in or near the eye, but there seems to be a great variation in the dose needed to produce this cataract and in the time of its onset.

<div align="center">DISCUSSION</div>

Mr. T. Harrison Butler: I remember seeing a tumor with a pedicle, which on biopsy was reported as hemangioma, a variety of nevus. Certain sprays of growth were seen going into the cornea, and I thought that feature suggested malignancy. The patient was sent for treatment to the Radium Institute, and after two applications of radium the condition was cured. In another case the view of the late Treacher Collins and other authorities was that the growth was epithelioma. It was pigmented but was only slightly raised above the general surface, though it implicated the cornea to a distance of several millimeters. It is about twelve years since I first saw the patient. She had several applications of radium at the Radium Institute, and during the twelve years there has not been a suggestion of recurrence.

Mr. James asked me to see a man who had been subjected to roentgen treatment for a long time in order to cure an extensive nevoid condition on his face and in whom a typical radiation cataract had developed. So far as I can see, there is no difference between the cataract caused by the roentgen rays and that caused by radium, if those agents are injudiciously used over a considerable time. I think the risk of causing cataract by using the doses needed in these cases is infinitesimal.

Mr. A. D. Griffith: The fear in connection with these cases is the risk of causing radiation cataract. Has Mr. King used much heavier doses of radium in these cases for a shorter period of time, say, 30 millicuries for half an hour, and is this not much less likely to cause

cataract? In one case in which the latter ensued the first thing I noticed was a change in the refraction, and six months later cataract began.

MR. E. F. KING: Technically, the method I have been using is as simple as that carried out at the Radium Institute; it can be repeated when necessary. As to the employment of a heavier dose for a shorter time, I was guided in the matter of procedure by the advice of Mr. Stanford Cade; if I observe a similar case in the future I shall put the point before him and ask his views. His feeling about the lens was that with the doses of radium used in these cases there was little risk of producing cataract.

GERMAN OPHTHALMOLOGICAL SOCIETY

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DOCENT M. BÜCKLERS, M.D., Tübingen, *Reporter*

TRANSLATION BY PERCY FRIDENBERG, M.D., New York

Second Scientific Session

Monday, July 6, 1936, 3 p. m.

W. MEISNER, M.D., Cologne, *Chairman*

DISCUSSION OF REPORT IV

DR. O. MARCHESANI, Munich: As to the interpretation of the term blindness, I think it corresponds largely to that of incapacity (*Invalidität*) of visual origin. It is clear that we wish to prevent the progeny not only of blind subjects but of incapable subjects as well. If we have recourse to the conception of incapability, this offers the advantage of a concept which is familiar to all ophthalmologists. The statement "One must anticipate that with the greatest probability in the following generations there will be hereditarily diseased subjects" is not to be understood in the sense that these probabilities must necessarily be determined in each individual case. This would lead to perfunctory or intentionally motivated decisions. On the contrary, the commentary attached to the general provisions of the sterilization law state definitely that this great probability is presented in all cases of hereditary disease which is known to be transmitted according to either the dominant or the recessive mode. The recognition of a disease of this nature is sufficient. In our opinion, zonular cataract frequently originates in factors of the environment (*Umwelteinflüsse*). Slitlamp studies frequently show definitely at just what period of intra-uterine life or at what age the damage was done. There is to my mind no contradiction between the old view that this disease is due to rickets and the prevalent opinion that tetany is the etiologic factor of prime importance. For it is well known that in

the healing stage of rickets there is a disturbance of metabolism which results in a general spasmophilia. So rachitis is not so much the cause of zonular cataract as it is an indicator. The question arises whether we are not to envisage the cases of hereditary zonular cataract and those of zonular cataract which has developed on a basis of spasmophilia from a uniform standpoint. According to von Pfaundler—and subsequent research in cases of identical twins has supported this view—the tendency (*Anlage*) to rickets is itself hereditary (rachitic diathesis).

It is probable that this *Anlage* may be inherited in varying degrees, quantitatively. In the cases of manifestly hereditary zonular cataract the tendency alone would be decisive, whereas in the cases of other types of zonular cataract an additional factor, that of upset metabolism, is requisite for the manifestation of the disease.

Dr. Wissmann, Wiesbaden: Does the practical ophthalmologist merely have to report patients for sterilization, or is it part of his function in the evaluation of the condition to make a detailed scientific report and, in a way; to place at the disposal of the court (*Erbgericht*) his own opinion as an expert to serve as a basis for the decision?

Dr. Engelking, Heidelberg: It would be a mistake to minimize the difficulties which may present themselves in the practical carrying out of the sterilization statutes. A warning is in place that the diagnosis is not to be considered the only, or even the predominant, leading datum. For instance, not every patient with retinitis pigmentosa should be certified for sterilization without further consideration. There are, without doubt, families in which this condition is hereditary and hereditable (*vererbbar*) and in which the members through several generations have maintained fairly good vision for life. Just as little does every patient with total color blindness deserve to be sterilized, as there are patients with this condition, as I can report from my own experience, who have perfectly good sight and are practically undamaged. Some congenital hemeralopes are quite capable of gainful occupation. These, too, in my opinion, need not be sterilized. Another difficulty is presented by the fact that certain diseases appear now as hereditary and again as nonhereditary. This applies, for example, to microphthalmos, which may occur in connection with microcephaly by external traumatism, or by damage to the fetus, such as that caused by the roentgen rays. The same is true of coloboma in association with defective sight. In these and analogous disorders it is my opinion that sterilization should be called for only when positive proof of the hereditary character of the condition is forthcoming, reserve being maintained in cases in which the condition occurs sporadically (*isoliert*).

Dr. W. Clausen, Halle: The enormous value of the sterilization statutes can be fully grasped only from the standpoint of general medicine and racial hygiene, not merely from that of ophthalmology alone. Ninety per cent of those to be sterilized are subjects with a congenital mental defect, and in such cases a decision surely ought not to be difficult. Of the remaining 10 per cent, those who are to be sterilized on account of hereditary ocular disease represent only a very small fraction. I have sharply defined the concept of practical blindness as far as it applies and is possible for the application of the law. It corresponds,

one might say, with a visual disability so extreme that incapability (*Invalidität*) is caused by it. Each case must be considered separately and evaluated from every angle. It goes without saying that heredity must be definitely established and that the report of examination and the history must be definite and complete and the diagnosis correct. It is highly probable that more intensive research in the immediate future will tend to show that glioma is hereditary in many more cases than has been assumed heretofore. In the past the question of possible inheritance was not looked into thoroughly, as glioma was not considered hereditary until quite recently. In regard to total color blindness, I have called attention to the fact that it is generally associated with nystagmus and visual defect of the highest degree. Only such types of total color blindness and not those in which vision was approximately normal were considered.

INDIVIDUAL PAPERS

I. Parenchymatous Keratitis. Dr. J. Blaickner, Salzburg.

On the basis of the observation that patients with interstitial keratitis present, in greater percental frequency than patients of the same age with other conditions, anomalies in the development of the thyroid gland which are verified by inspection and palpation, the basal metabolic rates for 34 patients with this condition, who were observed in 1933-1936, were determined according to the method of Krogh and evaluated in relation to the body weight and the body size in accordance with the standards in Benedikt's tables. In 3 of these cases the determination of the basal metabolic rate could not be carried out on account of non-cooperation and fear on the part of the patient, but in all the other cases reliable curves for breathing were obtained. The values found varied between + 10 and — 10 per cent in 15 cases, but it is to be noted that by far the greater number of these values lay below the zero point. However, on account of the mathematical limit of error of about 10 per cent, which is the general allowance, this entire group is reckoned as normal. The figures were between — 10 and — 20 per cent in 6 cases, between — 20 and — 30 per cent in 2 cases and between — 30 and — 40 per cent in 5 cases, whereas there were only 4 cases in which the values were between + 10 and + 36 per cent. Naturally, an attempt was made to check up these figures with those for controls. Determinations in 10 control cases gave the following figures: In 4 cases the values were between — 10 and + 10 per cent; in the other 6 the values were between + 10 and + 32 per cent. Of the patients with parenchymatous keratitis, 11 were between 4 and 10 years of age, 13 were between 11 and 20 and 9 were between 21 and 30, and 1 patient, a woman, was 36 years old. Eighteen of the patients were males and 16 females. In his material the reader was unable to find any relation between the low basal metabolic rate and the sex or the age of the subject. On the other hand, the few patients with a high metabolic rate were all under 15 years of age. The results of these investigations, then, with all necessary critical reserve, show a definite preponderance of negative values, i. e., minus figures; but in this connection it must be borne in mind that even possibly

very slight diminution in the basal metabolic rate, which in a given case cannot be accurately determined on account of the comparatively wide limits of error inherent in the method, may be decisive if one considers the highly complicated cell metabolism in the cornea—a nonvascular tissue—particularly when one is dealing with a pathologically altered or destroyed cell life. On the basis of the results of these clinical tests, treatment with thyroxin was added to, and combined with, the invariably and thoroughly carried out antisyphilitic therapy. One or two ampules were given weekly ("i. m." [intramuscularly?—P. H. F.]), depending on the degree of lowering of the basal metabolic rate. No shortening of the course of the disease could be noted, but there was a definite change in the clinical course.

If the patient had been treated from the very beginning with thyroxin in addition to the antisyphilitic therapy, it was striking that in all the condition frequently and regularly ran the same course, which remained unchanged for a long time. This condition was characterized by Igersheimer as the type of parenchymatous keratitis with posterior or deep deposits, and separate, scattered foci of opacity in the deepest layers of the cornea, immediately in front of Descemet's membrane. These posterior deposits often persisted obstinately for a long time, but the final result was, nevertheless, a favorable one, as the middle layers remained unscathed throughout a wide extent. The reader is of the opinion that more importance should be attached to disturbances in the metabolism of the thyroid gland as an etiologic factor in parenchymatous keratitis, although that does not indicate, by any means, that he denies the importance of anaphylaxis. The main factor must, of course, be seen in the active syphilis effective somewhere in the organism, possibly on the posterior surface of the cornea.

II. KERATOCONJUNCTIVITIS SICCA—PARTIAL SYMPTOM OF A MAJOR SYNDROME. DR. HENRIK SJÖGREN, Jönköping, Sweden.

As to the symptomatology of this disorder, the reader refers to a monograph by him, published in 1933 in the *Acta Ophthalmologica*. General examination revealed the following details: 1. The sedimentation (*Senkungs*) reaction showed increased figures in all the cases. 2. The blood picture was, with few exceptions, characterized by lymphocytosis; in isolated cases there was a low degree of eosinophilia and in a larger number a medium degree of anemia. 3. The body temperature was subfebrile for a longer or a shorter period in all the patients, 11 in number, who were examined. These reactions indicate that one is dealing with a chronic infection. The pathologic changes in the conjunctiva consisted in a high degree of atrophy of the epithelium. The lacrimal glands as well as the glands of the nose, mouth, larynx and pharynx, were markedly sclerosed, and these glandular changes came on earlier than the lesions of the mucous membrances. Keratoconjunctivitis sicca is, accordingly, a partial symptom of a larger symptom complex which depends on infection and which when fully developed presents, in addition, xerostomia, rhinopharyngolaryngitis sicca and chronic polyarthritis. The various symptoms described as combined in this syndrome were formerly, as far as they were known, considered as separate, individual disorders, and there were only speculation and theory as to their

etiology. It is possible, of course, that there are various forms of atrophic rhinitis, differing in etiology. But these investigations prove that in one form, at least, they may occur as indications of a larger symptom complex, that in such cases they depend on an endogenous infection which attacks the glands and that the diminished secretion caused by the disease of the glands brings about the pathologic changes in the mucous membranes.

DISCUSSION

Dr. W. Löhlein, Berlin: In Berlin we have made it a principle, long ago, to apply the bengal rose (fluorescein) test in every case of obstinate bacterial inflammation of the conjunctiva, especially in patients at the menopause, and have found keratoconjunctivitis sicca frequently in combination with one or another of the symptoms which Dr. Sjögren has summed up in his monograph. I should like to discuss in detail a typical case in which the subject showed the entire symptom complex in its fullest and completest expression, as it indicates how severely the patient—a woman, as is almost always the case in this condition—was plagued.

The patient, aged 55, the wife of a colleague, had been treated in every way, but without effect, for an annoying and obstinate conjunctival catarrh with stringy secretion and varying opacities and plaques (*Flächenbildungen*) in the cornea. She suffered extremely from unbearable itching, and from blinking, as she could never keep her eyes open for any length of time, and had very annoying and painful sensations after reading or using the eyes for other work at short range for even a short time. The test with fluorescein was positive, giving the characteristic picture, and on closer investigation we learned that the patient had had parotitis in childhood and that this had recurred several times. Besides, she had had dryness of the buccal mucous membrane for years and could not eat without drinking at the same time. The teeth had become brittle, and sensitive swellings had developed about the finger joints. Without going into the details of the general physical examination, which gave essentially negative results, it may be mentioned that there was great relief from contact glasses, which were worn and well tolerated for three or four hours and in this space of time completely relieved the annoying symptoms, as in the moist chamber the epithelial lesions disappeared, and with them insufferable itching. It was very instructive to note the results of the fluorescein test before and after the wearing of the contact glasses. In the first case the lower two thirds of the conjunctiva bulbi and of the cornea were closely sown with reddish points in the epithelium. After the contact lens was removed the test was absolutely negative for the entire area which had been covered and protected by the glass. For the exposed portions beyond this area the test was still positive and showed the old superficial lesions. For severe conditions of this sort, accordingly, one can advise the careful selection of a contact lens.

Dr. Marchesani, Munich: Dr. Weve has informed me that conjunctivitis sicca is favorably influenced by instillations of fibrolysin solution. The results of the use of this medicament in one case of this condition in our own experience confirms his therapeutic observation.

III. THE AGING OF THE LENS. HANS KARL MÜLLER, M.D., Basel, Switzerland.

The lens is, above all organs of the body, that which has the greatest independence, as is shown particularly by the organ-specific action of lens albumin parenterally administered. Senile cataract, i. e., the death of the lens due to age, can, accordingly, be nothing else than the death of any given living organism as the result of age. In order to construct a basis for a rational medicamentous therapy for cataract it is essential to determine first the processes which are important for the life of the lens and whether they are within the lens itself or in its more or less immediate neighborhood. Then one must study the development and changes in these processes with age. The central point of therapy for cataract is the recognition of the dependence of the functional development of the lens on age conditions. On this principle, closer studies were made of glycolysis in the lens. These showed that aqueous solutions of lens substance have the property of changing added hexosediphosphoric acid into triosephosphoric acid. When the former chemical was added, the lenticular property of forming esters of organic substances with phosphoric acid was proved. Furthermore, if glycerin phosphate is added to aqueous solution of lens substance, phosphorus is split off. In lenses of old cattle both the aforementioned reactions were present only in a slight degree when the experiment was conducted under similar conditions. However, the property of splitting organic phosphoric acid compounds seems to be maintained at its former level. These and other senile changes explain why the lenses of aging animals contain decidedly less carbohydrate phosphates than those of younger cattle. With Lohmann's hydrolysis process it can be shown that especially the difficultly hydrolyzable fraction of the carbohydrates diminishes with age. This is probably to be attributed to a gradual disappearance of the phosphoglyceric acid. This finding also explains the diminution in the vitamin C content of the aging lens. It was shown that rabbits which had been intoxicated for fourteen days with large doses of phlorhizin had only a small amount of vitamin C (15.9 mg. per hundred cubic centimeters) in their lenses, while the lenses of control animals fed with sodium fluoride for ten days had a very high content (37.6 mg.). Normal animals of the same age showed a vitamin C content of 25.9 mg. in the lens. These reactions to phlorhizin and sodium fluoride, respectively, make it appear probable that phosphoglyceric acid is a precursor of vitamin C. These chemical investigations throw light on the functional changes which take place in the lens in the course of aging and if carried further may lead the way to a future medicamentous therapy of opacities of the lens and cataract.

<div align="center">DISCUSSION</div>

DR. JESS, Leipzig: It is fortunate that we have in our midst research scientists who are willing to devote themselves to the difficult investiga-

tions in the borderland of physiologic chemistry and, of late, of the vitamins and hormones. Twenty years ago when I began to busy myself with physiologic chemistry a prominent clinician informed me that these questions were of slight concern for ophthalmologists. There is no doubt that this old view was a mistaken one, and that if we are to get any nearer to the solution of the problem of medicamentous treatment of lenticular opacities we must, unconditionally, make use of the ways and means placed at our disposal by physiologic chemistry and all the exact methods of natural science. I need only refer to the papers announced for the next few days on the formation of cataract, epithelial bodies, and an irradiated ergosterol preparation which may well be of special significance in the question of the medicamentous effect on opacities of the lens.

DR. REINHARD BRAUN, Rostock: Dr. Müller has called attention to the organ specificity of lens albumin as cited, in his time, by Uhlenhuth, and has laid stress on this factor as one of the most important bases of modern research on the chemistry of the lens. I cannot let this statement pass unchallenged and uncontradicted. Numerous experiments have proved to my entire satisfaction that, at least in regard to the anaphylactic components, it is quite permissible to entertain doubts as to the organ specificity of lens albumin. Lens matter alone is quite capable of producing toxic effects which in their further development have an extraordinary similarity to anaphylactic reactions.

DR. HANS KARL MÜLLER, Basel, Switzerland: In answer to Dr. Braun's remarks, the peculiar individuality of the lens does not depend on the organ-specific action of parenterally administered lens albumin alone. As a matter of fact, even if the organ specificity of lens albumin is not complete, one has still to consider the anatomic and physiologic independence of the lens. This is definitely expressed in the fact that the lens has no blood vessels and is not in direct connection in any way with the central nervous system. Furthermore, although the nutrient fluid of the lens, i. e., the aqueous, contains barely perceptible traces of albumin, the lens is able to organize its chemical composition, probably by metabolic processes, in such a way that it finally has an albumin content of 35 per cent. From this and similar examples one may recognize beyond a doubt that the lens has an independent faculty of biologic development far beyond that of any other organ in the body.

IV. THE PROBLEM OF ELECTRIC CATARACT. DR. W. COMBERG, Rostock.

The research in this study was carried out in collaboration with Dr. K. Quest. In cases of injury by lightning and/or currents of high intensity, as well as in experimental studies of electric cataract, it has been shown that very often a severe iridocyclitis complicates the clinical picture. Up to the present time the question to be settled is whether the cataract is essentially due to the passage of the high voltage current through the lens and its capsule, or whether, on the other hand, it is caused secondarily by the severe inflammation of the uveal tract. The reader attempted to solve this question by making a change in the experimental conditions from those in the earlier usual course of research. This consisted in applying the electrical discharge in the region of the

orbital margin of rabbits or placing the electrode directly on the orbital margin and passing the current through the eye, essentially, in the equatorial direction. The reader placed one electrode on the middle of the cornea, using as a second electrode a clip, which was inserted behind the globe; the latter was kept dislocated and proptosed during the experiment. With this arrangement the anterior pole of the lens gets the strongest effect of the current, while the middle portions of the iris and the ciliary body get comparatively little. In the majority of the cases, after subjection of the lens to the current, an opacity developed at the anterior pole, directly underneath the capsule or in the anterior cortex and in the neighboring deeper layers. Even with this new arrangement of the experiment irritation due to iridocyclitis was frequently noted. The opacities of the lens tended to remain stationary, or they increased but slightly in the course of the following few months. The rapid development of the opacities of the lens in the subcapsular region, which had been exposed to the most direct and intense current, indicates strongly that one is dealing without much doubt with a direct action of the electric current alone. Another fact which seems to prove that there was no essential cooperation of an iridocyclitis in these experiments is the clinical observation that in those cases in which uveitis was noted, later progress in the opacities of the lens was as slight as in the cases in which iritic irritation had been minimal.

V. A Fourth Type of Phakomatosis. Dr. J. van der Hoeve, Leyden, Netherlands.

The term phakomatosis was proposed by the reader as a collective concept, derived from φακός, taken to indicate a birth mark or congenital nevus, for syndromes presenting symptoms of a congenital *Anlage*, in which heredity is a factor, for example, the syndromes of Bourneville (tuberous sclerosis of the convolutions of the brain), von Recklinghausen (multiple neurofibromatosis) and von Hipple and Lindau, all of which consist in spots or flecks (phakoi), tumor-like malformations (phakomas) and actual neoplasms (phakoblastomas) complicated by congenital anomalies. In accordance with Albrecht's classification of the origin and genesis of tumors, phakomas may be put into the following categories: (1) choristomas, which are malformations arising from cells scattered in a kind of diaspora far from their usual site, and (2) hamartomas, which develop from autochthonous cells usually found, normally, in the same location, from unused cells which have remained in situ, from embryonal interstitial connective tissue, and so on. The term hamartosis is rejected, because this includes only a part of the symptoms. A picture of a phakoma of the retina in a case of phakomatosis (von Recklinghausen) was sent to the reader by Pascheff, and in the microscopic sections of an eye of one of the patients with Bourneville's phakomatosis which the reader reported in 1921 not less than four retinal phakomas were found. These probably developed by way of internal metastasis via the vitreous, as the reader showed in 1923. A diagram was presented in which the phakomatoses are divided into phakoi, phakomas, and phakoblastomas. The first group comprises congenital birth marks of the following types: (1) phakoi with nevus cells, (2) homotopic phakoi and (3) heterotopic phakoi. The phako-

blastomas, which are true neoplasms, may be benign or malignant. The phakomas, finally, which are tumor-like formations of anomalous histologic and biologic provenance, include: (1) choristomas, which originate from disseminated germinal cells, e. g., hypernephroma of the kidney; (2) hamartomas, which originate from varieties of cells which are normally found in situ, for instance, adenoma sebaceum, (3) phakomas which originate from remnants of unused or quasi-superfluous cells,· e. g., neurogliocytoma, and (4) phakomas which originate from embryonal interstitial connective tissue, for instance, glioma as a starting point for syringomyelia. Further, in a phakoma of the nerve head and in a retinal phakoma in a case of Bourneville's phakomatosis, calcified spots were found. The reader placed in the category of the fourth type of phakomatosis Sturge-Weber's disease, which has the following symptom complex: nevus vinosus, buphthalmos, vascular anomalies and angiomatosis of the choroid, calcifications in the brain, epilepsy, idiocy, paralyses, acromegaly, adiposity, atrophy of the brain, angiomatous proliferations in the pia mater and other conditions. He reported a new case, that of a girl aged 3½ years with a very extensive port wine mark of the face, hemiplegia, mental retardation, glioma of the retina, calcifications in the brain and crossed paralysis (on the right, cerebral, and on the left, cerebellar). Deposits of calcium were found only in the brain, not in the cerebellum; accordingly, deposits of calcium and atrophy are, as Krabbe also claimed, independent of one another. The calcium is not bound to the blood vessels, as Bergstrand and others believed. In this brain, which was studied by Prof. B. Brouwer, the calcifications were quite independent of the vessels. The reader pointed out that the plates of calcification in this brain and those in the intra-ocular phakomas in cases of Bourneville's phakomatosis are deceptively similar, even to the point of being indistinguishable, while similar calcifications are also found in the tumors of the ependyma of the ventricles which are generally present in Bourneville's disease. It would appear that this variety of calcification is a symptom quite independent of the phakomatoses. Gliomas are seen in all four varieties of phakomatosis; viz., in the brain in Bourneville-Recklinghausen and/or von Hippel-Lindau disease, and in the retina in the syndrome of Sturge-Weber. There are transitional manifestations from one variety of phakomatosis to another, buphthalmos being seen in Recklinghausen and Sturge-Weber's disease, and retinal phakomas in all forms. There are suggestions of, and analogies (*Anklänge*) with, Hand-Schüller-Christian's disease and with Recklinghausen's bone disease, in the latter case probably owing to the presence of a phakoma in the parathyroid glands. None of the phakomas is limited to any one definite germinal layer. The reader laid stress on the great practical importance of a study of the phakomatoses for increasing knowledge of the structure of the brain and of the pathogenesis of tumors. He also hailed the advance of the knowledge of this topic in the last twenty years, the increased efficiency of therapeutics and the importance of diagnosing the so-called formes frustes (abortive or atypical forms).

DISCUSSION

DR. E. SEIDEL, Jena: Although Dr. van der Hoeve took heredity into consideration in his interesting remarks, I should like to inquire

whether he has carried out any intensive and extensive studies of a genetic nature in this comparatively large series of what are, after all, relatively rare clinical conditions. The subject is of particular interest because in the cases of retinal angiomatosis which I have observed inheritance and/or familial occurrence was noted repeatedly. Thus, in my original communication on the subject to this society (*Ber. u. d. Versamml. d. deutsch. ophth. Gesellsch.,* 1912, p. 335), in which I was the first to call attention to the cerebral symptoms or complications of angiomatosis retinae, I recorded the fact that the brother of my patient with angiomatosis retinae had died, like the latter, as a result of cerebellar cyst. Later I reported (*Ber. u. d. Versamml. d. deutsch. ophth. Gesellsch.,* 1932, p. 535) that both children of the first patient with angiomatosis of the retina who was seen by von Hippel, a daughter and a son, were affected by angiomatosis retinae in each eye. The daughter died as a result of cerebral complications, so I was able to make a microscopic study of the eyes, each of which showed multiple retinal tumors, which I demonstrated at the time at Leipzig. Recently I observed a third case of this disease with familial occurrence, at Jena. The father had an angiomatosis of the retina associated with cerebral symptoms. The family history showed that some years previously a daughter had been operated on elsewhere for a cerebellar cyst and had died subsequently.

Dr. FLEISCHER, Erlangen: In addition to the specific and characteristic tumor-like changes in the retina, for which, however, one must search carefully, as they may be very fine and minute in some cases, one notes other lesions. Especially in the peripheral portions of the fundus, one finds small, round, white atrophic foci which at first give the impression of being old atrophic postinflammatory changes but which are undoubtedly to be considered as atrophic foci on account of the characteristic changes in the retina. They are certainly of definite importance for the diagnosis of tuberous sclerosis.

Dr. J. VAN DER HOEVE, Leyden, Netherlands: The little white foci in the retina in cases of Bourneville's disease may be of various provenance: They may be small areas of choroiditis, possibly degenerated phakomas, or they may be phakomas in the incipient stage, which may later on develop and grow. It is probable that there are many patients with phakomatosis in institutions, hospitals for persons with mental diseases, and so on, but to us the ambulant patients are more important, those who are going about and who show few striking symptoms, if any, those with the formes frustes, such as the patient already mentioned, the woman belonging to a family in which phakoma was present, who showed only one symptom, viz., a small retinal tumor, while, on the other hand, one of her children was seriously ill. The factor of heredity plays a rôle in all phakomas, and the problems it presents are very difficult. The mother of this girl had asked me whether her daughter's condition was hereditary and whether the latter could marry, as she did not want the daughter to go through the miseries that she herself had endured. After consulting with the neurologists I felt that, although there was a very small tumor-like formation in the retina, I did not have the right to hinder marriage. Up to the present time we know practically nothing about the hereditary character of this disease. If several children of this woman show this disease it will

be, to some degree, my fault. And this consideration again makes me realize how serious the problems are which the questions of heredity now put up to German ophthalmologists. When I came here it was my firm conviction that it is not permissible to sterilize on account of ocular disease alone, and that conviction has not been shaken by all that I have learned yesterday and today from the discussions as well as from the papers. In my opinion, we should exercise the greatest possible caution in this matter, for various reasons. For me, the supreme law for the physician is *primam non nocere,* and one does not know how seriously one injures the patients physically and, above all, psychically; one cannot foresee how great the influence will be of such a procedure, which is of itself not serious but which may well have a serious effect on an organism already damaged by the disease. My second reason for objection is that at present we know far too little about this entire momentous subject to be entitled to go ahead ruthlessly. We must surely recognize and admit that genetics as a science is still in its infancy, however highly we value much of the work that has been done in this field. Very little, on the whole, has been definitely established. We do not even know how sex is transmitted as a heritage. We say that the female has two x-chromosomes and that the male has only one and in addition one y-chromosome. Incidentally, the only possibility of inheriting a sex-linked variation is that the variation be linked to the y-chromosome, and then, surely, only the male sex would be liable to it. From the hypothesis of x-chromosomes and y-chromosomes it follows that just as many males as females would have to be born. However, we know that figures still show that more males are born, so that the theory has not been definitely proved. We may, possibly, get proof from a study of variations obtained from the sex chromosome. For that reason I agree heartily with Franceschetti that it is unquestionably essential to investigate thoroughly the family tree of all color-blind subjects. Yesterday there was mention of a manifest heterozygosity in Leber's disease, which we know to be a sex-linked recessive disorder. This is, in my opinion, a contradiction in terms. In the most extensive family tree of dominant heredity thus far studied, viz., that of Jean Nougaret in hemeralopia, the figures do not correspond at all with the theory, and an attempt is made to explain this discrepancy by the assumption that perhaps the spermatozoa which were afflicted with this variation heritage were less fruitful than others. The path of inheritance is entirely unknown in the case of glioma as yet. In regard to aniridia, which is a typically dominant inheritance, many cases occur as solitary, i. e., nonfamilial. There is much talk of so-called irregular dominance and so on, which probably means merely that the facts and the theory do not fit. We do not know yet, for example, what the percentage of cases of hereditary blindness is. As long as our ignorance is so great, I think we should exercise the greatest reserve as to active procedures and interfere only in cases in which such action is absolutely necessary. And as I am convinced that such interference is rarely, if ever, necessary for patients who have ocular anomalies only, my opinion is now, as it was previously, that, considering the present state of our knowledge, obligation to sterilize on account of ocular defects alone is not permissible.

VI. Diabetic Retinitis. Dr. C. Mylius, Hamburg.

In the course of the last four years 362 of the patients admitted to the hospital for persons with diabetes were also examined ophthalmologically. In 59 of these we found retinal changes which stand in a causal connection with the systemic disease. Those patients were intentionally disregarded in this analysis who had retinal lesions of another sort, such as retinitis proliferans, thrombosis of the central retinal vein or embolism of the central retinal artery, in which perhaps some dependence on the existing diabetes might have been thought of. In all previous papers which have had to do with diabetic retinitis, the factor of high blood pressure has been featured prominently, again and again. In fact, Volhard stated quite definitely—seven years ago, it is true—that he did not know of any cases of diabetic retinitis without increased vascular tension. If we scan the clinical histories of our large group of patients with normal retinas and compare them with those of the group with retinitic changes we must admit on the basis of this parallel that the blood pressure is essentially lower in the first group than in the second. For this several factors are authoritative: 1. In the group with retinal changes the patients were almost without exception elderly subjects—the two youngest in this group were 37 years old. 2. It cannot be explained away that, even if we compare like age categories, the blood pressure in the second group, i. e., that of patients with positive pathologic retinal findings, is, on the average, higher than in the group with normal fundi. Naturally, that alone does not prove anything, for the moment, as to the etiologic significance of high blood pressure in the pathogenesis of diabetic retinitis. We must now call attention to the fact that, in spite of the generally higher blood pressure in the patients with lesions of the fundus, there were no less than 15 in whom the blood pressure was quite normal or even definitely lowered. So it was evident that these, above all, were the patients who called for our especial attention. A most intensive and careful clinical study, with use of all methods of examination, demonstrated that of these 15 patients 8 would have to be excluded, as they were useless for purposes of comparison on account of various intercurrent conditions, such as cardiac decompensation and other severe complications, including carcinoma, tuberculosis, marked emaciation or subsequently increasing values for the blood pressure. We think we can state definitely, however, that in the remaining 7 patients the existing lower levels of the blood pressure are actually to be considered those which we must take into account. Further, it was found that in all these cases we were dealing with type 1 and/or 2 of the categories which Grafe had set up on a basis of Hirschberg's definitions, viz., a condition characterized by fine white stippling and large white plaques, with more or less numerous sharply outlined hemorrhages, particularly at the posterior pole, absence of retinal edema, and normal disks with clearly defined, regular margins. Examination of the vascular system of the retina in these cases showed without exception slight irregularity and variations (*Schwankungen*) in the caliber of, and a somewhat accentuated light streak on, the arteries and, without any essential exceptions, a change in the relation of the diameter of the arteries to that of the veins, of about 2 : 4. Marked changes in the walls of the vessels were completely missing in these cases, whereas they are very frequently found in group

3 of Grafe's schematic division. These 7 patients were also found to be uniformly free from albuminuria and had, as far as it could be tested, normal renal function. In type 3 of Grafe's category, just referred to, there are always renal complications, and these are made evident not only by the presence of albumin in the urine and marked increase of the blood pressure but regularly, also, in the ophthalmoscopic picture by the appearance of cloudy margins of the disk, retinal edema and marked changes in the walls of the vessels to the point of complete obstruction. We are, accordingly, convinced that we cannot consider these 7 cases in which the blood pressure was low as exceptions which prove the rule. On the contrary, in these instances we are actually dealing with early cases of diabetic retinal disease which must lead us to look at the so often misapplied symptom of increased blood pressure as what it really is, viz., a frequent manifestation running parallel with the retinal disease, one which may be missing in the early stages of that disease and which, accordingly, has nothing to do with the pathogenesis of the retinitis. Further observations tended to corroborate this view. First, in a patient with a perfectly normal blood pressure and normal eyes a bilateral retinitis developed in the course of five years. Second, in 2 cases of retinal lesions which did not belong to the group of the 7 related cases just cited the rise in the blood pressure was observed quite later than the retinal lesions, and certainly did not precede their appearance. As circulatory changes similar to those which we judge to be at the bottom of retinal vascular disease are found in other organs, the increased blood pressure proves to be, as it may be, for the greater part, in our cases, a phenomenon of somatic compensation and regulation. This rejection of increased blood pressure as a systemic, symptomatic factor to be considered in the pathogenesis of retinitis diabetica must not be interpreted and applied incorrectly. For if, on the basis of our observations, we look on the rise in the blood pressure in many of our cases as a compensatory phenomenon, it is logical to conclude that there must be conditions calling for and requiring such compensation, and these are, above all, vascular and circulatory disturbances. The frequency and practical clinical significance and importance of these circulatory complications did not become clear in its fullest extent until after the discovery of insulin. In former times most diabetic patients died of the disease itself, i. e., in coma. Today Joslin's dictum holds true that the diabetic patient dies not of the disease but of the complications, and among these are to be reckoned the circulatory and vascular complications particularly. Even at the present time the change from a diet rich in fat and albumin to one markedly free from these ingredients and rich in carbohydrates has not been carried out generally and consistently. It will have to be worked out gradually. It lays less stress on normalizing the blood sugar than on elimination of ketone bodies, the aim being to prevent the formation of these dangerous substances by maintaining sufficient glycogen reserves in the organism. ·

(To be continued)

Book Reviews

L'oculistica di Antonio Scarpa e due secoli di storia. By G. Ovio. Two Volumes. Cloth. Pp. 2,296, with 202 figures in the text and 48 plates. Naples: Casa Editrice Libraria V. Idelson, 1935-1936.

Scarpa's name has always been associated with anatomy and surgery. Few know, however, that the "surgeon of Pavia," as Scarpa was called, like his teacher, Morgagni, was much interested in ophthalmology and that in 1801 he published his first book, "The Principal Diseases of the Eye." His clear style, accurate observation, clinical judgment and well balanced application of the principles of pathology known in his day made him a leader at the beginning of the past century.

This masterly work ran into many editions and was translated into German, Dutch, Spanish and English. It became a popular textbook on ophthalmology, in spite of the fact that many books on the subject in various languages were already in existence. About the middle of the past century the book was supplanted by more up-to-date and extensive treatises, and it was almost forgotten.

Professor Ovio, of the University of Rome, has done a service in bringing Scarpa's book on the diseases of the eye to the attention of ophthalmologists. He has taken Scarpa's original work as the main theme and has added to it a history of ophthalmology covering a period ranging from one hundred years before Scarpa's time up to the present.

Professor Ovio has divided his two volumes into two parts. The first part, which is rather short, is dedicated to the general pathologic and therapeutic conceptions of the period during which Scarpa lived; the second part, which is far more voluminous, covers Scarpa's work and is divided into twenty-two chapters.

The chapter dealing with the lacrimal apparatus, although not the longest in the book, is no doubt one of the most interesting. It is a masterpiece of anatomic description and clinical observation. The ingenious use of the so-called "Scarpa's nail" for lacrimal fistulas is described at length. This chapter is rich in the opinions of other ancient writers and of more modern authors and is profusely illustrated, giving the reader a complete comparative study of the subject from its obscure origin up to the present. No doubt even the well qualified plastic surgeon is much impressed to read of Scarpa's criteria on blepharoplasty and other surgical procedures on the lids. Although Scarpa was a skilful operator, he did not use Daviel's operation for extraction of cataract. Ovio comments that Scarpa did not follow this method; he preferred depression of the lens into the vitreous for so-called hard cataract, while he advised laceration of the anterior capsule for soft cataract. He was living in an age in which anesthesia and antisepsis were unknown. He had perhaps seen so many tragic sequels of hazardous extractions that he had become conservative.

His observation on staphyloma, with a consideration of its differentiation from keratoconus, is impressive and is worthy of the pains that Ovio takes in amplifying it with numerous historical and anatomicopathologic annotations. Tumors of the orbit are treated at length.

Scarpa, having been primarily a surgeon and a pupil of Morgagni, was expected to discuss such a topic fully.

In contrast to the topics mentioned, the subject of amaurosis is not very clearly explained. Still one must remember it was about fifty years after Scarpa's book was published that Helmoltz gave ophthalmologists the means of examining the fundus oculi.

Professor Ovio's two volumes are extremely interesting, being enriched with numerous plates representing various ocular conditions, and instruments used at the time of Scarpa, as well as before and after his time. The author has compiled a bibliography of some two thousand references, ancient and modern, which should be an excellent aid to the student of ophthalmology in his critical historical researches.

Ophthalmologists everywhere owe a debt of gratitude to Professor Ovio for these two valuable volumes, which should be in every library of works on ophthalmology.

G. BONACCOLTO.

Ophthalmoscopy, Retinoscopy and Refraction; with a New Chapter on Orthoptics. By W. A. Fisher, M.D., F.A.C.S. Fourth revised edition. Price, $2. Pp. 210, with 240 illustrations, including 24 colored plates. Chicago: H. G. Adair Printing Co., 1937.

The present edition of this volume has a new chapter of thirty pages with a bibliography of sixty-one references on the orthoptic treatment of strabismus. The method of Cantonnet is described at length, and there are outlines of the procedures used by Dobson, Peter, Guibor, Gifford and Cameron. Statistics are given as to the results obtained by various workers.

The remainder of the volume is similar to the earlier editions. One notes a great deal of unnecessary repetition throughout the text. Also, the treatment recommended for many of the pathologic conditions described in the sections on diseases of the retina, choroid and optic nerve is too briefly described. In the reviewer's opinion, therapy should have been discussed more fully or should not have been considered in the volume.

W. F. DUGGAN.

Nursing in Diseases of the Eye, Ear, Nose and Throat as Practiced at the Manhattan Eye, Ear and Throat Hospital, New York. Sixth edition. Price, $2.25. Pp. 288, with 93 illustrations. Philadelphia: W. B. Saunders Company, 1937.

In this edition, the sixth, the part on the eye, written by Dr. David Webster, has been revised and brought up to date. In the eighty pages devoted to the eye, the anatomic and physiologic features and the diseases of the eye are described. Then follow a discussion of the common remedies and a chapter on eversion of the lid, retractors, drops, solutions, ointments and salves. Contagious diseases of the eye, the nurse's duties at operations and instruments and appliances for the eye are the subjects of the final chapters.

A valuable part of the book is the chapters on antiseptics, sterilization and the nurse and her duties.

The text is clear, and the illustrations are well selected, so this small manual should serve as a useful guide for nurses.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6ᵉ.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

INTERNATIONAL OPHTHALMOLOGIC CONGRESS

Secretary: Dr. E. Marx, Costzeedijk 316 Rotterdam, Netherlands.
Place: Cairo. Time: Dec. 8-14, 1937.

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. P. E. H. Adams, 6 Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

GERMAN OPHTHALMOLOGICAL SOCIETY

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. T. E. Ashdown Carr, 34 Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3 Midan Soliman Pasha, Cairo.
Time: March 1938.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. Gordon M. Holmes, 9 Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27 Weymouth St., London, W. 1.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Time: July 8-10, 1937.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.

Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.

Place: Lindley'a 4, Warszawa.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31 East Southernhay, Exeter, England.

Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris, 7è.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm, Sweden.

Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.

Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.

Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.

Place: San Francisco. Time: June 13-17, 1938.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Lee W. Dean, Washington University Medical School, St. Louis.

Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

Place: Palmer House, Chicago. Time: Oct. 10-15, 1937.

American Ophthalmological Society

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.

Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.

Place: Hot Springs, Va.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.

Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

Place: New York. Time: Oct. 6-8, 1937.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.

Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

New England Ophthalmological Society

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Rd., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time:
8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. William Donoher, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: San Francisco. Time: June 22-25, 1938.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. W. Howe, 740 St. Helens Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except
June, July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501—7th St., Rockford, Ill.
Place: . Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of
each month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Andre Cortopassi, 703 Second National Bank Bldg.,
Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each
month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. J. H. Judd, 107 S. 17th St., Omaha, Neb.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.
Place: New Orleans. Time: Nov. 30-Dec. 1-3, 1937.

Southwestern Michigan Triological Society

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand
Rapids, Mich.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Place: Indiana, Pa. Time: Oct. 21, 1937.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all
members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the
month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Charles T. Flynn, 41 Trumbull St., New Haven.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Place: New London. Time: February 1938.

Eye, Ear, Nose and Throat Club of Georgia
President: Dr. John King, Thomasville, Ga.
Secretary-Treasurer: Dr. Mason Baird, 384 Peachtree St., Atlanta, Ga.
Place: Augusta. Time: May 1938.

Indiana Academy of Ophthalmology and Oto-Laryngology
President: Dr. E. E. Holland, 51 S. 8th St., Richmond.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: April 6, 1938.

Iowa Academy of Ophthalmology and Oto-Laryngology
President: Dr. James A. Downing, 406—6th Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines. Time: Nov. 18, 1937.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society
President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: New Orleans. Time: Spring, 1938.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology
Chairman: Dr. F. N. Smith, Grand Rapids Clinic, Grand Rapids.
Secretary: Dr. Dewey R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology
President: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology
President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology
Chairman: Dr. Oram Kline, 414 Cooper St., Camden.
Secretary: Dr. James S. Shipman, 542 Cooper St., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section
Chairman: Dr. Marvin F. Jones, 121 E. 60th St., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: May 10, 1938.

North Carolina Eye, Ear, Nose and Throat Society
President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology
President: Dr. H. Rosenberger, 221—5th St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514—6th St., Valley City.
Place: Bismarck. Time: May 1938.

Oregon Academy of Ophthalmology and Oto-Laryngology
President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. George Truluck, Orangeburg.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia. Time: Nov. 9, 1937.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Kate Savage Zerfoss, 165—8th Ave. N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.
Place: Nashville. Time: April 12-13, 1938.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. M. H. Hood, 505 Washington St., Portsmouth.
Secretary-Treasurer: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. James A. Fisher, 501 Grand Ave., Asbury Park, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, 106 S. Main St., Akron.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Secretary: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Thomas D. Allen, 122 S. Michigan Blvd., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Webb Chamberlin, 7405 Detroit Ave., Cleveland.
Secretary: Dr. Walter H. Rieger, Hanna Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, 25 Prospect Ave., N. W., Cleveland.
Secretary: Dr. L. G. Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. C. E. Silbernagle, 247 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. M. Johnson, 327 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. A. W. Davisson, City National Bank Bldg., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Lester H. Quinn, 4105 Live Oak, Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. J. D. Carroll, 102—3rd St., Troy, N. Y.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. L. Allen, 1215 Walker Ave., Houston, Texas
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. C. P. Clark, 24 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. O. S. Gilliland, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. F. C. Hertzog, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Arthur M. Zinkham, 815 Connecticut Ave., Washington.
Secretary: Dr. E. J. Cummings, 1835 I St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Secretary-Treasurer: Dr. Fowler Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. A. J. Young, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Ophthalmological Society of the University of Pittsburgh

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

Passaic-Bergen Ophthalmological Club

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Raymond A. Lower, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto, Canada.
Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 Eye St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

FATTY DEGENERATION OF THE CORNEA

(NEUTRAL AND LIPOID)

LIEUTENANT-COLONEL R. E. WRIGHT, C.I.E., I.M.S.
Professor of Ophthalmology, Madras Medical College

MADRAS, INDIA

In the ARCHIVES for May 1936 I[1] discussed two cases of corneal degeneration. One was definitely an instance of fatty degeneration, as proved by the demonstration of predominant neutral fat in histopathologic preparations. The other (case 1 in the article referred to) was regarded as a possible example of calcareous degeneration by reason of certain associated features, e. g., recurring inflammatory foci and hypercalcemia. This surmise has proved incorrect, and as I have since had the opportunity of investigating the change in the tissues in this case it is of interest to record the result.

The patient, Mr. J., on leaving the Government Ophthalmic Hospital on Feb. 13, 1935, returned to Malaya. Vision in each eye was within the normal limits. Owing to failure of postal communication, my report as to the nature of the condition and my suggestion that our respective records should be amalgamated and published did not reach Dr. Viswalingam, who had sent the patient to me. Consequently notes were published separately, mine, as has already been cited, and Dr. Viswalingam's.[2] In the latter article the patient's history was briefly sketched, and it was noted that after his return to Kuala Lumpur from Madras the left eye had to be removed, as it was blind and painful. The specimen was sent to London for pathologic investigation, and the report is quoted in full. The diagnosis made from the appearance was "sclerosing keratitis profunda." On February 13 I had suggested keratoplasty as the most hopeful line of treatment when both eyes became blind, and so, when the remaining eye lost vision, owing to the extension of the yellow infiltration throughout the whole cornea, Dr.

To be read in continuation of "Degeneration of the Cornea, Calcareous (?) and Fatty," published in the May 1936 issue of the ARCHIVES, page 803.

1. Wright, R. E.: Degeneration of the Cornea, Calcareous (?) and Fatty, Arch. Ophth. **15**:803 (May) 1936.

2. Viswalingam, A.: A Case of Sclerosing Keratitis Profunda, Brit. J. Ophth. **20**:449 (Aug.) 1936.

Viswalingam sent the patient back to me for corneal grafting. The patient arrived in Madras on July 18, 1936.

FURTHER NOTES ON CASE

Ophthalmic Examination.—The right cornea was completely infiltrated by yellow material with the exception of a small semi-opaque area near the limbus in the superior nasal quadrant. The corneal epithelium was smooth. The eyeball at this time looked very congested, but the patient said that only occasionally did he have these attacks of redness. He was taking large doses of sodium salicylate (30 grains [2 Gm.] four times daily) and considered that it controlled them. He still had recurrent inflammatory foci, which were present in the epitrochlear region, the metatarsophalangeal joint of the great toe and near the upper part of the tendo Achillis on each side, but they were not so definite as formerly. While the patient was under observation for a few days the sclera showed changes varying from pallor to intense redness. When in the pallid state it was observed to be definitely thinned out in the region of the ora serrata. The patient was reported to have had slight rises of tension during the congestive attacks in the past year. Apparently there was a difference in tension between the congested state and the more pallid state, but the rise was not to an abnormal extent, since, even in congestion the tension as measured with the Bailliart tonometer was below the average normal. The reason for these congestive attacks could not be determined. The patient had used pilocarpine for their relief, but it is doubtful whether this drug influenced the condition.

On examination with the corneal microscope it was perfectly clear that there were iridescent plates of some sort (not needles) in the middle layers of the substantia propria.

Examination of the Blood.—The blood calcium content was somewhat high, since the patient had given up the calcium-free diet which I had formerly advised. The blood cholesterol content was high, the figure being 294.2 mg. per hundred cubic centimeters of plasma. The fatty acid content of the blood was 370.8 mg. per hundred cubic centimeters of plasma; the serum calcium content was 13.6 mg. per hundred cubic centimeters, and the serum phosphate content 2.84 mg. A complete roentgen examination of the skeleton showed no change in the bones since the patient's visit of more than a year previously.

Captain T. W. Barnard, director of the Barnard Institute of Radiology, Madras, and Dr. M. J. S. Pillai, the medical superintendent, made these examinations.

Operation.—A keratoplasty was undertaken in the hope of giving the patient some vision, since this had dropped to perception of movements of the hand close to the face.

On July 25 a graft 7 mm. in diameter was taken from a donor of the same blood group and placed in an aperture 7 mm. in diameter made with a trephine in the center of the patient's cornea. It was seen on removing the disk from the patient that the cornea was at least one and one-half times the usual thickness. The chamber was shallow. There was an incipient cataractous condition of the lens; the iris looked normal, and the pupil was contracted. The graft was retained in place by three double waxed threads placed from limbus to limbus at angles of 60 degrees to each other, making six spokes radiating from the center of the graft, in accordance with my usual technic. The graft took well in spite of the discrepancy in thickness; the pupil was kept contracted with pilocarpine. There was some delay in the formation of the chamber, and anterior synechiae with the junction of the graft formed in several positions. The stitches were removed on the fifth day after the operation, and on the tenth day the graft was clear and

the patient had vision of 2/60. It was considered advisable to free the synechiae. This was done on August 13. The operation proved to be a mistake, as some blood clot formed in the pupil and diminished the vision. The freeing of the synechiae also caused a small detachment of Descemet's membrane, which started a haziness in the graft, with some vascularization. It looked as if this might determine permanent opacification of the graft, so that the prognosis for sight was bad. (Extraction of cataract would have been required, of course, in a short time had the graft kept clear.) In view of the recurring attacks of congestion
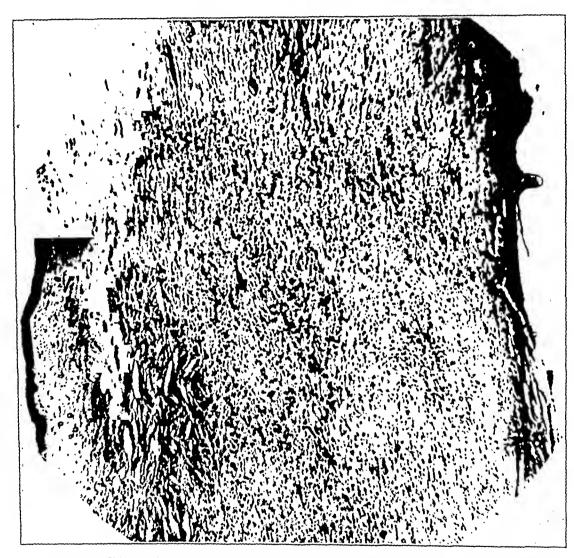
Fig. 1.—Photomicrograph of a section of the cornea of Mr. J., showing lipoid change associated with the deposition of plates of cholesterol.

and the thinning out of the sclera, it seemed highly probable that a staphylomatous condition of the sclera would eventually take place. The cornea was bad soil on which to plant a graft, although in the first instance there was a promise of some success. On August 31, when the patient was allowed to go home, his vision had definitely improved, and he could tell whether the hand was open or closed at 3 meters. He could count fingers at 50 cm. He was depressed at the result of the freeing of the synechiae but pleased to see at all.

Histopathologic Examination.—The disk of the cornea was immediately subjected to histopathologic investigation by Dr. T. Bhaskara Menon, pathologist of

the Stanley Medical School, Madras. His report confirmed the slit lamp observations at the time of this second visit, namely, that there were crystalline plates in the substantia propria. At the time of the patient's first visit the appearance was more suggestive of needle-like crystals than of plates, but as cholesterol may occur as either needles or plates, possibly the form of the deposit may have altered. The report, which follows, speaks for itself.

"I have examined the specimen of the cornea both by frozen sections and after paraffin embedding. Histologically the slides show that the surface epithelium is almost normal. Bowman's membrane is unaffected, except that it is separated in places from the epithelium. The substantia propria just beneath shows irregular oval slits which become more marked deeper down. These are spaces left by some crystalline substance which has been dissolved out. Round these slits the fibers of connective tissue are swollen and slightly hyaline, with compressed nuclei. Here and there round and oval vacuoles can be made out in the fibers. Deeper down, cellular infiltration becomes marked; the cells are mononuclear and eosinophile, with a few polymorphonuclears, all having a perivascular distribution; here the slits left by the crystalline deposit are more irregular. This cellular infiltration is most marked in the deeper layers. Frozen sections, stained and unstained, show that the crystalline deposits are surrounded by fine globules of neutral fat; these take the sudan III stain; the crystals do not take the stain. When teased out on a slide and examined they appear as characteristic rectangular plates with broken edges typical of cholesterol. They give the color reaction with acetic anhydride and sulfuric acid. I cannot find any trace of calcium deposit by von Kossa's stain. With osmic acid, black globules of fat can also be demonstrated in close relation to the crystals. Some are collected close to Descemet's membrane."

Figure 1 shows some of the changes just described.

COMMENT

It was obvious as the result of the second investigation of this case that my former notion as to its nature was incorrect. Even though the patient had hypercalcemia, attacks associated with inflammatory foci (the eye being also affected) and, in one of the nodes, an excess of tissue calcium, the corneal deposits were certainly not calcareous. The clinical picture might at first have suggested a gouty process, but this is ruled out by various findings. The abundance of cholesterol in the cornea suggests a third group of metabolic derangements, and this is supported by the hypercholesteremia. Is it possible that the inflammatory foci were associated with the deposition of lipoid? If so, the general condition would fall into the group of lipoidoses associated with defective lipoid metabolism, e. g., xanthomatosis and the syndromes associated with the names of Schüller and Christian, Gaucher, and Niemann and Pick. The general picture need not, however, concern one at the moment; there is hardly enough evidence to discuss it usefully. The corneal condition, on the other hand, is most interesting, as it affords a good example of one variety of lipoid degeneration or infiltration, namely, that associated with the deposition of plates of cholesterol. Although to the naked eye the appearance suggested a neutral fatty change, clinically rather like that in case 2 in my article

already cited, in case 1 there were histopathologic changes markedly different from those in case 2. In case 2 there was much more evidence of neutral fat, although mixtures of fatty acids and a few crystalline needles (of doubtful composition but not calcium) were present. In case 1 there was little evidence of neutral fat, but there was definite deposition of plates of cholesterol in spaces between the lamellae of the substantia propria. The clefts where the plates have disappeared are a marked feature of the histopathologic preparations. The tissue reaction was to a considerable extent associated with the deposition of the plates and was in part a foreign body reaction somewhat similar to the tissue reaction in the xanthomas. Both cases are, however, examples of those of a group of corneal degenerations which come under the generalized title of fatty degeneration or, better, perhaps, lipin degeneration. It may be helpful to think of this degeneration in two subgroups: (*A*) neutral fatty degeneration of the cornea (in which neutral fat predominates) and (*B*) lipoid degeneration of the cornea (including deposits of cholesterol, both crystalline (plates or needles) and amorphous cholesteride (such as coarse cholesterol, fatty acid esters, and finer intracellular or intercellular deposits, e. g., those of arcus senilis).

Cases of the *A* type are rare; in fact, fatty degeneration of any part of the globe associated with a predominant neutral fat change is rare. When fatty degeneration occurs in the globe the mode is ordinarily lipoidal, just as in certain other parts of the body, e. g., the aorta. I need not digress here in order to theorize on this theme.

Cases of the *B* type are not rare; for instance, arcus senilis is an everyday sight. In case 1 the condition was evidently a clinical variety of this subgroup; in the syndrome assumed it is probably uncommon, although in the degenerations of old corneal lesions the deposition of plates of cholesterol or needles is hardly so rare as one might judge from the infrequent references in the literature. One sees lipoid degeneration also from time to time as a yellow infiltrative process in keratoconjunctivitis eczematosa, trachoma and the interstitial keratitis of the granulomatous diseases.

I have recently seen a third clinical variety of corneal degeneration which falls into this subgroup. In this instance the color of the material was white, without a suggestion of yellow. The process was deep in the substantia propria, and there were deep vascularization, slight but definite interference with sensibility, and a completely clear narrow marginal ring, with the opacification starting where one ordinarily observes the arcus senilis. In fact, the condition might have been regarded as arcus senilis which had become excessive and irregular so that the advance of the opacity toward the center was not uniform

but was more intense in some quadrants than in others (fig. 2). The central portion of the cornea was not encroached on to any great extent but was faintly hazy. The interesting feature in the case was that with the corneal microscope the advancing edge of the denser areas showed definite needle-like crystals. (One cannot be sure of the exact nature of needle-like crystals, since substances other than cholesterol and fatty acid are met with in tissues in this form.) There were no associated symptoms of any importance—no jaundice and no glycosuria were present, and the blood chemistry was normal. This case recalled Dr. Parker Heath's [3] article in the ARCHIVES. It is not quite clear to me whether he wished to indicate a distinct clinical entity of metabolic etiology, in addition to suggesting that a number of corneal lesions which are now classified as separate entities might be grouped more accurately as lipin interstitial keratitis. There are, of course,
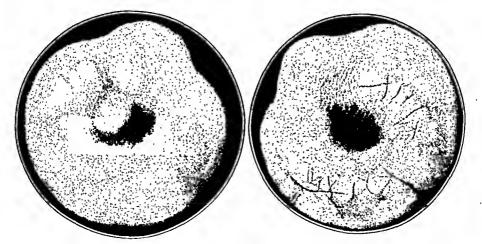
Fig. 2.—Corneal degenerative changes in the right eye (left) and the left eye (right) associated with the deposition of needle-like crystals (cholesterol?).

many corneal lesions in which lipin or lipoid degeneration takes place. It is of doubtful value to regroup them under a name signifying a common pathologic process since this does not help one to understand their etiology any better. For example, keratitis disciformis to me not only signifies a peculiar shape and distribution of a corneal lesion but in the vast majority of the cases which I see is one expression of a definite clinical entity, namely, the disease superficial punctate keratitis, which I have ventured to rename keratoconjunctivitis diversiformis et uveitis anterior. My associates and I have observed over thirteen thousand cases of this disease in Madras since 1928. We feel fairly certain that we know most of its clinical characters and think that we know its etiology. I have never seen the keratitis disciformis of this

3. Heath, Parker: Lipin Interstitial Keratitis, Arch. Ophth. **13**:614 (April) 1935.

particular disease result in lipin degeneration. I mention this point merely to illustrate how Dr. Parker Heath and I, although thinking along the same lines as regards the probable frequency and importance of lipoid degeneration of the cornea, might differ as regards the value of classifying corneal diseases by grouping them under the degenerative process concerned (which, after all, is no better a basis of classification than shape, color or any other feature) rather than according to etiology. My inclination would be to say that lipoid degeneration is a type of degeneration not infrequently met with in certain corneal conditions of unknown etiology now regarded as separate entities. I would also feel inclined to use the term lipoid degeneration rather than lipin degeneration, for lipin degeneration embraces neutral fatty degeneration, and this is rare, in my experience.

A fourth member of this subgroup is a distinct variety of primary lipoid corneal degeneration, which I[4] originally described in the *American Journal of Ophthalmology*. This is a relatively common clinical degeneration, formerly spoken of in this clinic as "corneal dystrophy of old persons" but now recognized to be a degeneration sui generis and not necessarily of old persons. Since my original description of this condition I have had an opportunity of doing a keratoplasty on an affected eye. The disk removed with the trephine was 11 mm. in diameter and afforded an opportunity of investigating corneal sections of whole thickness. The patient had corneal degeneration of each eye, and vision of each eye was perception of movements of the hand. The keratoplasty done on the left side was not a great success, since the large graft became somewhat steamy and there was a cataractous condition of the lens. Extraction of cataract would have given the patient a good practical visual improvement, probably of the order of ability to count fingers at a few meters, but the patient left the hospital, being advised to return for broad iridectomy and extraction of cataract. Vision of the left eye on discharge was ability to count fingers at 0.5 meters. Dr. T. Bhaskara Menon again conducted the histopathologic investigation, and his report is as follows:

The sections of the cornea show that the surface epithelium is thinned out and lost in part and abnormally thick elsewhere. In the substantia propria there is an infiltration of the connective tissue cells with small rounded droplets which fuse together to form larger globules and masses as described in the *American Journal of Ophthalmology*.[4]

In the center of the ulcerated area, the fibers of the substantia propria are frayed and broken, and the deposit has assumed the appearance of irregular homogeneous masses; the droplets, as well as the masses, take a reddish color with hematoxylin. They take a blue color with nile blue, suggesting fatty acid mixtures. There is no trace of neutral fat when frozen sections are stained with sudan III or osmic

4. Wright, R. E.: Interstitial Degeneration of the Cornea, Am. J. Ophth. **19**:413 (May) 1936.

acid. The globules and irregular masses have been identified as cholesterol or/ and cholesterol fatty-acid esters since they give a color reaction with acetic anhydride and sulfuric acid. I had this confirmed by Dr. A. S. Mannady Nayar, professor of biochemistry at the Madras Medical College. So the condition appears to be cholesterol steatosis, as originally suspected. As regards the site of commencement, I cannot be very definite. Descemet's membrane appears normal. In the case of Mr. J., already described—in which plates of cholesterol were asso-

Fig. 3.—Photomicrograph of a section of the cornea, showing changes due to primary lipoid or cholesteride degeneration.

ciated with marked cellular changes and capillary invasion of the cornea accompanied or succeeded the initial deposition of lipoid—we could trace the change from around the blood vessels in the substantia propria, but there is no cellular infiltration here.

Figure 3 shows some of the appearances described.

Another section shows that the epithelium outside the ulcerated area is entire and is slightly hyperplastic in places, forming papilliform buds projecting down. The deposit appears in places to be just underneath Bowman's membrane in the

substantia propria. There is distinct hyalinization of the connective tissue fibers of the substantia propria around the deposit. Slight infiltration by round cells can be made out round the vessels at one end.

This infiltration was noted near the limbus; one does not necessarily find vascularization in this type of degeneration, and the cornea surrounding a patch may be perfectly free from vessels when examined carefully with the corneal microscope.

Clinically and histopathologically this corneal malady is different from that in the two cases of subgroup B just discussed, although there is the common factor of lipoid degeneration. In the latter type of degeneration the microscopic appearance of the cornea is distinctive, examination revealing the amber-colored or oil-like masses and droplets. These are large toward the center of the patch, where the process is oldest and where the masses are big enough to push the epithelium forward in rounded elevations, here and there causing its thinning out and disappearance and leaving an eroded gap. It may be seen that between the elevations of thinned-out epithelium there are irregular areas of epithelial thickenings due to proliferation. Toward the edge of the patch the droplets become smaller until they fade away as colorless points constituting a gray haze at the periphery. This appearance is suggestive of a cloudy swelling which by analogy one might perhaps expect to precede intracellular fatty degeneration. On the other hand, it may be an intracellular edematous condition. We have as yet no definite evidence, since the available sections have not included the hazy areas. The histopathologic changes under low power magnification are such as those shown in figure 2 in my article in the *American Journal of Ophthalmology.*[4] The photomicrographs and drawing illustrating the present report, although poor, are somewhat more instructive. My recent investigation does not throw any light on the etiology of this condition, but since interstitial degeneration of the cornea—the name under which I described this condition in the *American Journal of Ophthalmology*—does not even indicate the nature of the histopathologic process, perhaps the name primary lipoid or cholesteride degeneration of the cornea might be more apt. At all events, the name will serve pro tempore to indicate a relatively common corneal degeneration seen here, and readily recognized, even in its earlier stages, by those who have observed a few cases. We have looked for correlations, without result. Apparently the lipoid metabolism may be normal, but the last patient examined, a woman (and the condition is not so common in women as in men) showed 200 mg. of blood cholesterol per hundred cubic centimeters. The sugar metabolism is not necessarily deranged.

Lipoid degeneration is, to be sure, a common postmortem appearance in this part of the world in the seats of election, viz., at the exits of the branches of the main arterial stem, but possibly not more so here than

elsewhere. The general resemblance between atheromatous degeneration of the great vessels and that form of degeneration just described suggests itself; the intimate nature of these processes is almost identical. The word atheromatous (or hyaline) has been used in referring to certain corneal degenerative conditions for a long time but not in the sense of primary degeneration. It would seem to be applied to changes taking place in old corneal lesions, and the yellow spots have been regarded as hyalin. Here let it be emphasized that the particular type of lipoid degeneration just described as cholesteride is not infrequently seen in old corneal opacities of various types. It is readily recognized by the brownish or amber-colored masses, or oil globule-like collections of cholesterol fatty-acid ester, which one sees from time to time when examining old corneal lesions with the slit lamp; but in such cases the condition is secondary to an initial corneal lesion.

One gets the impression that this is the commonest type of fatty degeneration of the cornea, whether primary or secondary, and it may be that it has been confused with hyaline degeneration. It is a matter of pathologic interest that when lipin degeneration does occur in the eyeball it most frequently assumes some variety of the lipoid mode of degeneration. One is familiar with it in the cornea, the lens, the vitreous, the retina and the optic nerve head, e. g., in the lens as cholesterin crystals and in the vitreous as crystals or the rounded bodies of Benson's asteroid hyalitis. In records of pathologic studies of diseases of the eye one seldom finds references to neutral fat except in the form of droplets associated with the predominant picture of lipoid change. That cholesterol often appears as a sequel to hemorrhage is true, but hemorrhage is not a necessary preliminary. In fact, lipoid degeneration of the cell protoplasm unassociated with hemorrhage is probably more common. It may perhaps be said of the eyeball, as of the aorta, that lipoid change is the mode of election when the concealed fat of the protoplasm of the cell is thrown out of solution by local degeneration or generalized interference with the lipin metabolism, e. g., in the lipoidoses.

That there may be types of lipoid degeneration other than those given in subgroup B goes without saying. This note is not intended to be an essay on the subject or, in fact, anything more than a record of some clinical observations with musings thereon which may interest others who have the leisure to collect data, make detailed observations and review the literature. I regret that lack of time has not permitted me to consult records bearing on the subject other than a small selection at hand.

HERPES ZOSTER ·OPHTHALMICUS COMPLICATED BY OPHTHALMOPLEGIA AND EXOPHTHALMOS

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The complications of herpes zoster ophthalmicus vary greatly among different patients. The major portion of the complications fall under four types: (1) keratitis, (2) iridocyclitis, (3) muscular palsies and (4) optic neuritis.

In about 50 per cent of all the cases the globe is affected, and in the majority of these (35 per cent of all cases) the cornea suffers.

Hutchinson stated that the globe is never affected except in cases in which the nasociliary nerve is affected, but exceptions to this rule have been reported. Thus, scleritis, superficial keratitis, keratitis disciformis, interstitial keratitis and other forms of deep keratitis are rather commonly observed. In most of these keratitides the iris is also affected, and both hypotension and hypertension have been recorded. Worster-Drought [1] stated that in 7 per cent of these cases the condition is associated with paralyses.

The third nerve is the most frequently involved, but it is unusual for the whole of the third nerve to be affected at the same time.

In a series of one hundred and fifty-eight patients Hunt [2] found twenty-four with paresis of the ocular muscles. He observed that the third nerve was affected in eighteen, the sixth nerve in five and the fourth nerve in one. Eighty of this group showed facial paralysis, and 64 per cent showed some associated paralysis in the nervous system.

Numerous cases of palsy of various nerves have been reported. Thus, palsy of the third nerve has been reported by Wallace,[3] Brissaud,[4] Zentmayer,[5] Fage,[6] Rosnoblet,[7] Gallois [8] and others; palsy of the fourth

1. Worster-Drought, C.: Brit. M. J. 1:970 (June 9) 1923.

2. Hunt, J. Ramsey: The Paralytic Complications of Herpes Zoster of the Cephalic Extremity, J. A. M. A. 53:1456 (Oct. 30) 1909.

3. Wallace, W. T.: Ophth. Rec. 20:119, 1911.

4. Brissaud, in Wilbrand, H., and Saenger, A.: Neurologie des Auges, Munich, J. F. Bergmann, 1921, vol. 8, p. 238.

5. Zentmayer, W.: Am. Med. 4:1007 (Dec. 27) 1902.

6. Fage: Rec. d'opht. 31:209, 1909.

7. Rosnoblet: Zentralbl. f. d. ges. Ophth. 15:426, 1925-1926.

8. Gallois: Clin. opht. 14:421, 1925.

nerve by Caspar [9] and Vogel; [10] palsy of the sixth nerve by Galezowski and Beauvois [11] and Carpenter, [12] and combinations of palsy of various nerves by numerous other authors.

Aubineau [13] reported a case of typical herpes zoster complicated by complete fixation of the globe and associated with mydriasis and no reaction of accommodation to light. The patient died of nephritis, and necropsy revealed thrombo-arteritis obliterans of the ophthalmic artery, especially of the ramifications to the gasserian ganglion. Further cases of total ophthalmoplegia were reported by Metz, [14] Velter, [15] Leplat, [16] Barrière, [17] Silcock [18] and Batignani. [19]

Batignani's case was that of a 66 year old woman with a negative Wassermann reaction in whom herpes zoster was complicated by secondary uveitis, mydriasis of the pupils and anesthesia of the cornea. In the course of two weeks this patient showed total ophthalmoplegia associated with moderate exophthalmos (16 mm. in the right eye and 19 mm. in the left). Both the ophthalmoplegia and the exophthalmos cleared up entirely in two months. Batignani attributed the total ophthalmoplegia to direct extension to the oculomotor extrinsic musculature, and the exophthalmos to atony of the extrinsic muscles, with resulting proptosis.

Exophthalmos as a complication of herpes zoster has been reported by only a few authors. G. ten Doesschate [20] recorded a case of herpes zoster ophthalmicus on the right in a man aged 52, which was complicated by marked exophthalmos and by a rise in tension to from 60 to 70 mm. of mercury. The exophthalmos was considered to be the cause of the hypertension, and a trephining was done, which lowered the tension, the exophthalmos remaining the same. The tension rose again in a few days but dropped to normal when the exophthalmos began to decrease. Ten Doesschate believed that the proptosis was the result of stimulation of the sympathetic fibers.

9. Caspar: Arch. f. Augenh. **48**:177, 1903.

10. Vogel, F. E.: Augenmuskellähmungen bei Herpes zoster ophthalmicus, Inaug. Dissert., Leipzig, E. Lehmann, 1912.

11. Galezowski, J., and Beauvois, A.: Rec. d'opht. **28**:654, 1906.

12. Carpenter, in Wilbrand, H., and Saenger, A.: Neurologie des Auges, Munich, J. F. Bergmann, 1921, vol. 8, p. 239.

13. Aubineau: Clin. opht. **7**:513, 1914-1915.

14. Metz: Ohio State M. J. **7**:330, 1902.

15. Velter: Arch. d'opht. **43**:634, 1926.

16. Leplat: Bull. Soc. belge d'opht., no. 63, 1931, p. 39.

17. Barrière, A. V.: Arch. urug. d. med., cir. y especialid. **3**:498, 1933.

18. Silcock, in Wood, Casey: American Encyclopedia of Ophthalmology, Chicago, Cleveland Press, 1913, vol. 8.

19. Batignani, A.: Boll. d'ocul. **13**:814, 1934.

20. Ten Doesschate, G.: Paper read at the general meeting of the Netherlands Ophthalmologic Society, December 1918.

In the discussion of ten Doesschate's case, Mulock, however, reported a case of herpes zoster ophthalmicus complicated by exophthalmos which followed periodontitis. He attributed the exophthalmos to collateral edema; the tension was normal.

Vialeix, Prosper-Veil and Isnel [21] described a case of herpes zoster of the right eye of a 66 year old patient, complicated by paralysis of the third and sixth nerves and associated with exophthalmos. The exophthalmos appeared gradually following the zoster. The Wassermann reaction was positive, and the exophthalmos was thought to be due to inflammation of retrobulbar tissue.

Voisin [22] related a case of the disease in a man aged 73 who on the first day of the disease presented exophthalmos, palsy of the abducens nerve and total oculomotor paralysis. The Wassermann reaction was negative for both the blood and the spinal fluid; no definite etiologic factors were found. Mobility returned in two months.

<div align="center">REPORT OF A CASE</div>

A. R., a Negro aged 40, was referred to the Cook County Hospital on June 30. 1934. He stated that three weeks previously he first noticed an eruption of blisters, which appeared over the right side of his forehead and nose. This condition became progressively worse, and three days later he first noticed a rather rapid protrusion of the right bulb. This protrusion increased in amount over the next one and a half weeks.

There was a history of chancre fifteen years previously and an "abscessed tooth" one week before the onset of the ocular trouble, but the latter infection had cleared up after three or four days, without medical aid.

Examination of the eyes showed vision of the right eye to be ability to count fingers at 2 feet (61 cm.) and vision of the left eye to be 0.8, and with the left eye the patient could read Jaeger's test type 1 at 16 inches (40.6 cm.). There was an eruption of vesicles, some of which had already dried and crusted and a few of which were secondarily infected, over the region of distribution of the first branch of the right trigeminal nerve, including the nasociliary branch, so that the lesions were found on the upper lid, on the forehead as far as the scalp and also on the nose. The eruption was sharply delimited at the midline.

The lids of the right eye were edematous, and the lower lid was ectropionized by a large roll of chemotic bulbar conjunctiva. The bulb was proptosed; the right eye showed exophthalmos of 32 mm.; the left eye, exophthalmos of 24 mm. (as tested by the Hertel exophthalmometer).

There was complete immobility of the globe. Corneal sensation was slightly diminished. There was marked ciliary injection, and the cornea was diffusely cloudy, with a clover-shaped superficial fluorescein-staining ulceration covering most of the central three fifths of the cornea. Deep infiltrates were also scattered throughout the cornea. The iris was muddy brown. The pupillary margin was adherent to the lens by dense posterior synechiae throughout almost its entire extent. The pupil measured 4.5 by 5 mm. and was irregular and fixed. The

21. Vialeix, V.; Prosper-Veil, and Isnel, R.: Ann. d'ocul. **162**:569, 1925.
22. Voisin, M. J.: Ann. d'ocul. **73**:820, 1936.

tension was normal on tactile pressure. The corneal clouding obscured all the details of the fundus. The left iris was brown; the pupil and disk were normal.

No focus of infection was found in the ears, nose or throat. The department of oral surgery reported the right lower third molar as a possible source of infection; this tooth was extracted on July 4. The remaining part of the physical examination gave essentially negative results.

The urine was normal; the Wassermann reaction was markedly positive.

Local treatment consisted of the instillation of a 1 per cent solution of atropine sulfate four times a day and treatment with atropine powder to break up the synechiae, together with the application of heat. General treatment was carried out with neoarsphenamine in doses of 0.6 Gm., given at weekly intervals. Solution of posterior pituitary did not relieve the pain, but this abated spontaneously three or four days after admission.

After two weeks the superficial infiltrates were epithelized, but otherwise the findings were the same as previously and remained so for another month.

Photograph of the patient, showing herpetic lesions.

On September 9 the exophthalmometer readings were 30 mm. for the right eye; 24 mm. for the left, and slight motion of the bulb was returning. Irradiation with the roentgen rays in several half-erythema doses was applied to the right orbital region to stimulate resolution. By November 4 almost complete range of ocular motions had returned, and the conjunctival roll had receded. Vision of the right eye was 4/200, and there was marked superficial vascularization extending for 2 mm. into the cornea from the limbus around its entire circumference. However, the deep opacities in the stroma showed little absorption. The exophthalmometer readings at this time were 26 mm. for the right eye and 24 mm. for the left. The details of the fundus were still obscured by the corneal condition.

Reexamination on Dec. 9, 1936, revealed vision of 0.3 in the right eye with glasses and vision of 1.2 in the left eye with glasses. The old scars from the herpetic eruption still remained. The ocular motility was normal. The superficial vascularization was the same as previously. Numerous diffusely scattered superficial nebular corneal opacities were present.

COMMENT

The attempt to determine the cause of the ophthalmoplegia and of the exophthalmos in these cases has elicited various explanations. As early as 1871 Wyss described numerous hemorrhages in muscle substance and ascribed the muscular paresis to thrombophlebitis. It appears that the inflammation involves not only the posterior root ganglions and the nerve fibers originating from these ganglions but also the skin, the mucous membrane and other structures supplied by these nerves.

Various authors have called attention to the increased prevalence of herpes zoster in syphilitic patients, and it is difficult to say whether the case reported in this paper was one of the epidemic type occurring in a syphilitic patient or one of symptomatic type due to syphilitic basilar meningitis. However, in this case, as in Voisin's case, the clinical appearance was that of inflammation of the retrobulbar tissue.

GLAUCOMA AT THE WILLS HOSPITAL
1926-1935

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The object of this work was to analyze all cases of glaucoma in which the condition was diagnosed as such in the clinical records of the Wills Hospital during 1926 to 1935, inclusive.

The term glaucoma is applied to a variety of conditions in which the common physical sign is pathologically increased intra-ocular pressure. Some forms of glaucoma may be considered as disease entities, but etiologically, pathologically and clinically they vary so much that they should not be considered as forms of the same disease. However, the present knowledge of the etiology is so meager that it appears better and certainly more practical to describe the various types of ocular hypertension generically by the name of glaucoma. Accordingly, no attempt was made (with the exception of cases of secondary glaucoma), to group the cases on the basis of etiology or pathology; they were simply listed under the diagnoses used for the purpose of filing in the hospital. These diagnoses were classified as follows:

    a. Primary glaucoma
        (1) Infantile
        (2) Juvenile
        (3) Acute congestive (uncompensated)
        (4) Chronic congestive (uncompensated)
        (5) Noncongestive or simple (or compensated)
    b. Secondary glaucoma, classified as to etiology

In the ten year period 1,876 records of patients with conditions diagnosed as glaucomatous were available, including records of patients seen both in the dispensary and in the wards. On the basis of a total of 242,533 patients admitted to the hospital, this represents an incidence of glaucoma of 0.78 per cent. Data for each patient were assembled from the records of both the dispensary and the house on an individual record card prepared for each patient.

The following items were listed in each record, an effort being made to obtain such information when possible.

1. Factors of age, sex, marriage status, occupation, race and nationality
2. History, including mode of onset, symptoms and previous treatment
3. Specific diagnoses, classified as:
    a. Primary glaucoma
        (1) Infantile
        (2) Juvenile
        (3) Acute congestive
        (4) Chronic congestive
        (5) Noncongestive or simple
    b. Secondary, classified as to etiology
4. State of refraction
5. Visual acuity, visual fields and intra-ocular tension
6. Local ocular findings, including the condition of the cornea, anterior chamber, iris and pupil, lens, nerve head and retina
7. General physical status of the patient, with reference to the presence of syphilis, arteriosclerosis, hypertension, cardiorenal disease, diabetes, arthritis, imbalance of the endocrine glands or of the sympathetic nervous system, etc.
8. Laboratory data
9. Treatment
    (a) Operative
    (b) Nonoperative
10. Subsequent course, as checked by visual acuity, fields, tension and development of complications (infection, cataract)
11. Present status, both physical and sociologic, of the patient, determined from hospital records and from follow-up study of the patients living in Philadelphia

Of the 1,876 patients, 768 were residing outside of Philadelphia. No attempt was made to contact these persons. Letters were written to 1,008 patients in Philadelphia in whose cases it was felt that further information would be desirable; 573, or 56.8 per cent, of the letters were returned by the post-office as undeliverable. The remaining 535 patients were visited in their homes by field workers to obtain further information about the status of the patients and to persuade them to visit the hospital for follow-up study. Of the latter group, 264 patients actually reported, of whom 147 were found to have true primary glaucoma.

It is pertinent here to compare the figure of 0.78 per cent for the incidence of glaucoma in a large ophthalmic hospital situated in a metropolitan area and drawing patients from a surrounding well populated territory with figures from various countries as given in the literature.

The data in table 1 were gathered from various sources and show the incidence of glaucoma in various countries of three continents.

The data in table 2, taken from Nelander's[1] report (1933), are more authentic, since the number of actual cases of ocular conditions reviewed and the number of cases of glaucoma found are given.

The general uniformity of these figures is striking and would indicate that glaucoma bears a fairly fixed ratio to the general population.

TABLE 1.—*Incidence of Glaucoma in Various Foreign Countries*

Country or City	Investigator	Year	Percentage
Asia			
Indo-China..........................	Bargy	1929	2.00
Turkestan..........................	Pokrowsky	1924	2.10
Siberia—Russia......................	Cykulenko	1930	1.40
Kasan—Siberia......................	Batarekukov	1929	2.50
India—Madras......................	R. E. Wright	1929	2.60
India—Madras......................	R. E. Wright	1930	2.00
India—Madras......................	R. E. Wright	1931	1.60
North China—Peiping...............	Pillat	1932	1.11
Japan.............................	Joshida	1927	0.86
China.............................	Pillat	1935	1.17
Africa			
Egypt.............................	McCallan	1922	1.75
Tunis—Zarkum......................	Cuénod and Nataf	1928	2.00
Europe			
Denmark...........................	Peters	....	2.40
Berlin.............................	Graef	....	1.27
Wiesbaden.........................	Peters	....	1.48
Vienna............................	Peters	....	1.26
Breslau............................	Peters	....	1.84
Göttingen..........................	Schmidt	....	0.40
Tübingen..........................	Schüssele	....	0.73
France.............................	Peters	....	1.30
Paris..............................	De Wecker	....	1.17
Portugal—Lisbon...................	da Gama Pinto	....	1.06
Greece—Athens....................	Bislis	....	1.25
Poland............................	Mikulinska	....	0.64

TABLE 2.—*Incidence of Glaucoma in Patients with Ocular Conditions (Nelander)*

Author	Number of Investigated Cases of Ocular Conditions	Cases of Glaucoma	Percentage
Ahlström..........................	109,621	1,208	1.10
Coppez............................	148,000	1,511	1.02
Schmidt-Rimpler...................	55,146	688	1.24

SECONDARY GLAUCOMA

Cases of secondary glaucoma comprise a distinct group and should not be classed and described as cases of glaucoma, for the condition is simply intra-ocular hypertension with attendant symptoms due to some more or less definable cause. It may vary from simple hypertension closely resembling mild primary glaucoma through all gradations to a violent burst of hypertension with all the characteristics of acute con-

1. Nelander: Acta ophth. **11**:370, 1933.

gestive glaucoma. All cases of intra-ocular hypertension should be studied with the purpose of determining whether they should be grouped with cases of primary glaucoma or with those of secondary glaucoma. On the basis of the data available in the hospital records an attempt was made to do this, and 413 cases were classified as secondary—22 per cent of the total series of cases of glaucoma. This is to be compared with Carvill's[2] figure of 40 per cent of cases of secondary glaucoma in her series of cases of glaucoma observed in the Massachusetts Eye and Ear Infirmary.

TABLE 3.—*Grouping of Cases of Secondary Glaucoma According to Etiology*

Cause	Number of Cases
Trauma	101
Cataract	67
Iritis	57
Uveitis-choroiditis	46
Keratitis, corneal ulcer	30
Iridocyclitis	27
Dislocated lens	13
Intra-ocular tumor	15
Retinal vascular lesions	12
Retinal detachment	3
Congenital defect of the iris	1
Postoperative secondary glaucoma	27
Unclassified	14

Table 3 indicates a grouping according to etiology. The individual groups in this table may be analyzed as follows:

Trauma.—This was found to be a factor in producing secondary glaucoma, either with or without perforating injury to the eyeball, in 101 cases. From the standpoint of this review it is significant only to list the forms and the eventuality of treatment. Thirty-five patients received only medical treatment. Operative procedures were performed on 45; the operations included the following:

Iridectomy	12
Paracentesis	4
Elliot trephine operation	5
Modified Lagrange operation	2
Enucleation	22

Two patients were found to have syphilis, which may or may not have been a factor. The end-results of treatment in this series are shown by the fact there was loss of vision of the affected eye in 35 per cent of the cases.

Cataract.—Swelling of the lens during the course of senile cataract or following the development of traumatic cataract caused secondary glaucoma in 67 cases. In 25 of these the condition was traumatic in

2. Carvill, M.: Tr. Am. Ophth. Soc. **30**:71, 1932.

origin; in 3 it occurred in association with complicated cataract, in 2 in association with diabetic cataract and in the remainder in association with cataract of the senile type. In 5 cases an Elliot trephine operation was necessary to reduce the tension, in addition to extraction of the lens. In this series enucleation was performed in 14 cases—a figure showing poor results in 21 per cent of the cases.

Iritis.—Glaucoma that developed during the course of disease of the iris was present in 57 cases; in 16 of these the patient had a positive Wassermann reaction and the condition was recorded as syphilitic in origin. Blindness resulted in 16 eyes—an incidence of 28 per cent.

Iridocyclitis.—The only causes determined for this condition were syphilis (1 case) and tuberculosis (1 case). Poor visual results occurred in 13 cases, or 49 per cent.

Choroiditis.—There were 46 cases in which choroiditis was a factor in causing secondary glaucoma. In 11 of these the condition was syphilitic and in 2 tuberculous. Loss of vision occurred in 6 eyes, or 13 per cent.

Disease of the Cornea (Keratitis, Corneal Ulcer).—In 30 cases secondary glaucoma was due to disease of the cornea; syphilis was a factor in 6. Loss of vision occurred in 6 eyes, or 20 per cent.

Dislocated Lens.—In 14 cases dislocation of the lens was a factor. In 10 of these cases the dislocation was traumatic. There was loss of vision in 9 cases, or 64 per cent.

Intra-Ocular Tumor.—Fifteen cases of secondary glaucoma due to intra-ocular tumor were listed, in 4 of which the growth was melanotic sarcoma of the choroid, in 1 spindle cell sarcoma and in 3 mixed cell sarcoma. Fourteen eyes were enucleated, 1 patient refusing operation.

Retinal Vascular Disease (12 cases).—Only 2 instances of secondary glaucoma following thrombosis of the central retinal vein were found—a surprisingly low incidence. There were 4 cases of intra-ocular hemorrhage secondary to vascular disease and 1 case of glaucoma following obstruction of the central retinal artery. In the remaining cases secondary glaucoma was associated with arteriosclerotic and hypertensive retinitis. Two patients were syphilitic.

Postoperative Secondary Glaucoma (27 cases).—In this group 12 patients had been previously operated on at other institutions and were seen in the Wills Hospital with the condition established. In 15 cases the condition was noted after operation in our own institution. The primary operative procedures and the number of cases were as follows:

Iridectomy	3
Capsulotomy	1
Discission	1
Extraction of cataract	22

This compares with a figure of 24 cases reported by Fox [3] (1936) from the Bellevue Hospital of New York for the period 1923-1934. In our series of 27 cases there was visual failure in 11—an incidence of 40 per cent.

Other Factors.—Retinal detachment was found associated with secondary glaucoma in 3 cases, with no apparent associated intra-ocular tumor. One case of secondary glaucoma in a patient with aniridia was observed. There were also 14 cases in which no diagnosis as to the etiology of the secondary rise in tension was available.

Comment.—On reviewing the series of cases of secondary glaucoma as a whole, it is found that trauma as a factor was present in 136 cases, an incidence of 32.9 per cent; syphilis was present in 31.5 per cent. The seriousness of the development of secondary glaucoma is made obvious by the figures for loss of vision, which range as high as 64 per cent in the cases of dislocated lens. If the cases of intra-ocular malignant growth requiring enucleation are omitted, the incidence of loss of vision in the series of cases of secondary glaucoma is 34 per cent.

It is our impression from reviewing these cases and also from a study of the records of the patients whose condition was diagnosed as primary glaucoma that in a goodly percentage of the latter the condition would be found actually to have its origin in some antecedent ocular disease. It would seem that the figure of 22 per cent for the incidence of secondary glaucoma is a minimum one, which could be increased by closer scrutiny of cases of glaucoma with a view to exposing cases of low grade, subthreshold uveitis acting to produce a secondary rise in tension.

CONGENITAL GLAUCOMA (BUPHTHALMOS)

Under this heading are classified cases of glaucoma due to prenatal defects in the development of the eye in which the filtrating mechanism is affected. Buphthalmos may be present at birth and progress rapidly, so that the condition is apparent and the patient is brought to the attention of the ophthalmologist during the early months of life or, in some instances, the anomaly may be of lesser degree and the symptoms of increased intra-ocular pressure may develop gradually. In the latter instances, patients are first brought to the clinic at varying ages. Occasionally this condition is overlooked in the early stage and is recognized so late that it is classified as juvenile glaucoma.

The consensus in the literature is that in the majority of these cases the condition is bilateral, that males are much more frequently affected

3. Fox, S. A.: Postoperative Glaucoma, Arch. Ophth. **16**:585 (Oct.) 1936.

than females, that heredity is a factor in the development of the disease and that other congenital anomalies of the eye may be present.

Buphthalmos is a comparatively infrequent disease. Gros [4] reported 8 cases, or 0.07 per cent, among 12,000 cases observed at the ophthalmic clinic of the Hôtel-Dieu, Paris. He collected altogether 45 instances. He found that the anomaly was noticed at or immediately after birth in 60 per cent of the cases, within the first year in 13 per cent, from the first to the third year in 17 per cent and later in 9 per cent. He found both eyes affected in 63.7 per cent. Of his cases, 61.2 per cent were found in boys and 38.8 per cent in girls.

Carvill [2] reported 4 cases of congenital glaucoma in 31,648 patients with ocular conditions examined during two years—an incidence of 0.01 per cent. All of the 4 patients were white boys. The condition was unilateral and was noticed shortly after birth. One patient presented a coloboma of the iris as an associated anomaly.

TABLE 4.—*Age at Which Patients with Congenital Glaucoma Were First Seen in the Clinic*

Age, Years	Number of Patients
Under 1	8
2 to 5	6
5 to 10	4
10 to 20	6
20 to 30	2
30 to 40	2

In our series 28 patients with congenital glaucoma were discovered— an incidence of 0.011 per cent. Of these, 20, or 71 per cent, were males, and 8, or 29 per cent, were females—which strongly bears out the opinion that the condition is sex-linked and is predominant in males. All the patients were of the white race. This is somewhat at variance with Zentmayer's [5] opinion that buphthalmos is more frequent in the Negro. Table 4 denotes the age at which the patients were first seen in the clinic. There were no definite data as to the influence of heredity in these cases.

In 3 of the patients, one eye alone was affected; in the remaining 25 the condition was bilateral.

The refractive state of the involved eyes could be determined in only 3 cases and in all these it was of a high myopic nature. This finding corresponds with the general opinion that myopic defects are relatively common in buphthalmos.

No positive Wassermann reaction was found in any of the cases, so the influence of congenital syphilis apparently did not play a part.

4. Gros: Etude sur l'hydrophtalmie ou glaucome infantile, Thèse de Paris, no. 237, 1897.

5. Zentmayer, W.: Ophth. Rec. **22**:25, 1913.

The physical characteristics of the buphthalmic eyes were in most cases typical of the condition, i. e., a large cornea, a deep anterior chamber, atrophy of the iris and excavation of the optic disk. In 2 instances the lens was dislocated, and in 1 case it was calcified; 1 patient had detachment of the retina. Vision was uniformly poor in most of the cases, ranging below 3/60.

Treatment.—As the patients included in this survey were taken from the services of nine surgeons, there were a variety of treatment and a lack of uniformity that make evaluation difficult. Eight patients received treatment with miotics; 6 were subjected to posterior sclerotomy at one time or another; in 3 cases an Elliot trephine operation was performed, and 4 eyes were enucleated.

The results for only 10 patients could be secured on follow-up study. Among these vision was maintained in only 3, and in 7 the visual result was poor. This is in keeping with the general opinion that the percentage of cases in which the treatment of congenital glaucoma with miotics is successful is low and that the prognosis is generally poor.

JUVENILE GLAUCOMA

The exact definition of juvenile glaucoma is difficult, and many authorities are at variance as to the prevalence of the condition as a pathologic or clinical entity. Again, writers use different standards as a criterion for making the diagnosis. Some consider all primary glaucoma occurring in persons under the age of 35 as juvenile glaucoma; some take 40 years as the upper limit, and others 20. Glaucoma which occurs as a result of congenital defects should be excluded from this definition. It would appear that juvenile glaucoma should be considered as a disease of younger patients in whose eyes changes have developed which predispose to or excite an attack of hypertension.

Carvill[2] included in her series of cases of juvenile glaucoma those cases of primary glaucoma occurring in persons between 5 and 40 years of age, excluding cases of buphthalmos. We have adopted this grouping and have attempted to classify the records similarly.

Accordingly, 20 cases of juvenile glaucoma are reported, in 12 of which the patients were females and in 8 males. This is at variance with Löhlein's[6] report that juvenile glaucoma occurs more frequently in males, although Haag[7] stated that the predominance of the disease in one sex was not well marked in his material.

Our patients ranged in age from 18 to 32 years; none of them had had any previous treatment. No hereditary influence could be demonstrated in any of these cases, although most authors have stated that

6. Löhlein, W.: Ber. ü. d. Versamml. d. ophth. Gesellsch. **12**:97, 1913.
7. Haag, C.: Klin. Monatsbl. f. Augenh. **19**:133, 1915.

heredity shows a more strongly marked influence in juvenile glaucoma than it does in the senile form of the disease.

The refractive state was determined for only 6 of these patients, 4 being myopes and 2 hyperopes. This may have some significance, as many authorities agree that myopia is found more often in persons with juvenile glaucoma than in those with senile glaucoma. In Haag's[7] series, 31 per cent of the patients were myopes, and 28 per cent were hyperopes. In Löhlein's[6] series, 50 per cent of the patients were myopic. In Carvill's[2] cases the condition was equally distributed between hyperopes and myopes.

Most of our patients were first seen when the condition was in an advanced stage, the vision being uniformly below 6/60. An associated anomaly was found in 1 case—bilateral coloboma of the iris. Löhlein found that 50 per cent of his patients had a congenital anomaly, as did Haag.

Three patients received treatment with miotics; basal iridectomy was performed on 5, the Elliot trephine operation on 3 and the Lagrange operation on 1; 1 eye was enucleated; the remaining patients were seen only once and did not return for treatment.

The results of therapy were unfavorable in most cases.

PRIMARY GLAUCOMA

The rest of the cases are considered cases of primary glaucoma; with the information available, an attempt was made to classify the forms of primary glaucoma in detail as: (1) congestive, inflammatory or non-compensated, or (2) simple, noncongestive or compensated. Type 1 was subdivided into: (a) the acute congestive form and (b) the chronic congestive form. The groups were assembled from the data noted in the records and the original diagnoses used for the purpose of filing, although changes were made in cases in which it was felt that the subsequent developments altered the diagnosis from the simple to the congestive type.

Under the heading of simple glaucoma we included those cases in which the patient complained of only visual loss without marked discomfort, with the development of increased intra-ocular pressure, typical changes in the field and excavation of the disk to any degree. Those cases in which there were definite symptoms of headache and ocular pain and local findings of ciliary stasis, corneal edema, a shallow anterior chamber, etc., were included in the group of cases of congestive glaucoma. These cases were divided into those of acute glaucoma and those of chronic glaucoma, depending on the degree and duration of the symptoms.

One thousand, four hundred and fifteen cases of primary glaucoma, including those of all types, were noted in this study, of which 392,

or 27.7 per cent, were cases of the congestive type, and 1,023, or 72.3 per cent, cases of the simple noncongestive type. Of the group of cases of congestive glaucoma, 161, or 42.9 per cent, were observed when the condition was in the acute stage, and in 231, or 58.1 per cent,

TABLE 5.—*Distribution of Cases of Primary Glaucoma According to Type, Age Group and Sex*

Age Group	Acute Congestive (11.4%)			Chronic Congestive (16.3%)			Simple (72.3%)			Total No. of Cases (Male 48.5%) (Female 51.5%)		
	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
40-49	7	10	17	13	15	28	58	67	125	78	92	170
50-59	37	20	57	28	37	65	120	122	242	185	179	364
60-69	26	28	54	35	53	88	188	195	383	249	276	525
70-79	9	21	30	18	25	43	124	118	242	151	164	315
80-89	1	2	3	4	3	7	19	12	31	24	17	51
	80	81	161	98	133	231	509	514	1,023	687	728	1,415

TABLE 6.—*Distribution of Cases of Primary Glaucoma According to Type, Age Group and Sex*

Age Group	Congestive (27.7%)			Simple (72.3%)		
	Male	Female	Total No.	Male	Female	Total No.
40-49	20	25	45	58	67	125
50-59	65	57	122	120	122	242
60-69	61	81	142	188	195	383
70-79	27	46	73	124	118	242
80-89	5	5	10	19	12	31
Total	178	214	392	509	514	1,023
Percentage	45.4	54.6		49.7	50.3	

TABLE 7.—*Summary of Distribution of Cases of Primary Glaucoma According to Type and Age Group*

Age Group	Acute Congestive		Chronic Congestive		All Cases of Congestive Type		Simple	
	No.	Percentage	No.	Percentage	No.	Percentage	No.	Percentage
40-49	17	10.5	28	12.1	45	11.5	125	12.2
50-59	57	35.4	65	28.1	122	31.1	242	23.6
60-69	54	33.5	88	38.0	142	36.2	383	37.4
70-79	30	18.6	43	18.6	73	18.7	242	23.6
80-89	3	1.9	7	3.2	10	2.5	31	3.2
	161		231		392		1,023	

the condition was considered to be in the chronic stage. Among the entire series of cases of glaucoma, those of acute congestive glaucoma comprised 11.4 per cent, those of chronic congestive glaucoma 16.3 per cent and those of chronic simple glaucoma 72.3 per cent. These figures, together with those showing the distribution of the cases according to age group and sex, are shown in tables 5, 6 and 7.

These figures may be compared with those found in similar studies reported in the literature. Carvill's [2] series included 52 per cent cases of noncongestive glaucoma and 46 per cent cases of congestive glau-
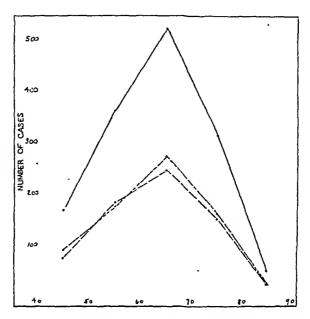
Chart 1.—Frequency of primary glaucoma at different periods of life. The long dash line indicates males; the short dash line, females, and the unbroken line, the total number.
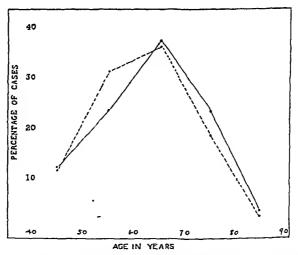
Chart 2.—Frequency of congestive and noncongestive glaucoma at different periods of life. The dash line indicates congestive glaucoma; the continuous line, noncongestive glaucoma.

coma—a greater incidence of the congestive type as compared with the incidence in our series. Weinstein,[8] in giving the results of a study

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8. Weinstein, P.: Monatschr. f. Augenh. **12**:794, 1934.

of 3,214 cases observed in thirty years at the University Hospital in Budapest, reported a vast predominance of cases of the congestive type, only 306 cases of simple glaucoma having been noted. Pillat,[9] in his series from China, made no distinction except to segregate the cases of acute glaucoma as an isolated group comprising 5 per cent of his series. Nelander,[1] in Uppsala, Sweden, in a series of 75 cases, found 6 instances of acute glaucoma, 7 of chronic congestive glaucoma and 62 of simple glaucoma. Trantas,[10] in discussing a series of 428 cases studied in Athens, Greece, reported 90.7 per cent cases of chronic simple glaucoma and 9.3 per cent cases of congestive glaucoma, half of the latter cases being instances of acute glaucoma.

Rohner,[11] who studied 200 cases in Switzerland, found an almost equal distribution of cases of these two types—48 per cent cases of simple glaucoma and 52 per cent cases of congestive glaucoma. In the

TABLE 8.—*Number of Cases of Simple and of Congestive Glaucoma Found in Previous Investigations (Rohner)*

Investigator	Year	Simple Glaucoma	Inflammatory Glaucoma
Illig	1921	311	95
Kraus	1899	155	78
Schmidt-Rimpler	1903	195	157
Neuberger	1894	136	134
Schüssele	1899	253	533
Basallino	1906	81	266
Jacobsohn	1903	30	115

latter group, 54 per cent of the patients showed acute phenomena at one time or another. Rohner also gave a table of results of previous investigations (table 8).

The considerable variance seen in these associated statistics from various countries is obvious and is undoubtedly due to the various criteria employed in classifying the congestive and noncongestive types. It would appear that with more intensive study of each case and with the employment of slit lamp examination more cases would fall into the group of cases of congestive glaucoma than have been reported. Moreover, such use of the corneal microscope in all cases would reveal changes, such as pigment dust on the iris or the capsule of the lens, cells in the anterior chamber, corneal deposits and other evidence of low grade uveitis, that would mark the case as one of the secondary type of glaucoma. Until such data are available, our cases must be classified, as they have been, merely on the basis of the history and the gross clinical findings.

9. Pillat, H.: Arch. f. Ophth. **139**:299, 1933.

10. Trantas, R.: Bull. et mém. Soc. franç. d'opht. **47**:277, 1934.

11. Rohner, M.: Schweiz. med. Wchnschr. **57**:780, 1927.

Age.—The factor of age is undoubtedly of the utmost importance in any statistical consideration of glaucoma; according to Elliot,[12] "Age takes precedence over all known causes of this disease." The distribution of cases during five decades of life for each diagnostic group and for the total group is shown in the tables and charts. The average age of the patients is 59.7 years for the females and 59.9 years for the males. According to census figures, the expectation of life for these persons is 14.1 years. This would indicate that one is dealing with a group of persons who will constitute a considerable proportion of the population for a substantial time and tends to emphasize the gravity of the situation in regard to glaucoma. Again, with the constantly increasing expectation of life and the survival of an older group of persons in the general population, the incidence of glaucoma will undoubtedly show an increase in the future.

In the series of 392 cases of congestive glaucoma, the greatest incidence, 36.2 per cent, was found for persons in the seventh decade of life. Similarly, in the group of cases of noncongestive glaucoma, in 37.4 per cent of the cases the condition occurred in the same period of life. Acute congestive glaucoma tends to occur somewhat earlier, the largest incidence, 35.4 per cent, being seen in the sixth decade of life, although chronic congestive glaucoma, like chronic simple glaucoma, appears most frequently in the seventh decade.

These figures agree substantially with those for Carvill's[2] series of patients, in 39.6 per cent of whom the condition occurred in the seventh decade, and with those for Haag's[7] series, in 31.8 per cent of whom it occurred at this time, although Priestley Smith's[13] series showed the greatest incidence by a small margin in the sixth decade, his figures being 29.3 per cent for the sixth decade and 29 per cent for the seventh decade. Weinstein's[8] series showed an incidence of 34.2 per cent in the age group of 55 to 64 in the cases of congestive glaucoma, although in his cases of noncongestive glaucoma the incidence for this age group tended to be somewhat lower, the greatest incidence being in the age group of 65 to 74. Pillat's[9] series in China showed an incidence of 34 per cent in the sixth decade. Nelander reported the greatest morbidity from glaucoma for persons in the age group of 70 to 79, Trantas for persons in the seventh decade of life and Rohner[11] for those in the second half of the sixth decade in all cases. In Rohner's cases inflammatory glaucoma tended to occur somewhat earlier than simple glaucoma.

Sex.—The influence of sex on the incidence of glaucoma has been accepted by practically all authors, the general consensus being that

12. Elliot, R. H.: Glaucoma, New York, Paul B. Hoeber, 1918.

13. Smith, Priestley: On the Pathology and Treatment of Glaucoma, London, J. & A. Churchill, 1891.

females suffer from primary glaucoma somewhat more frequently than males. Priestley Smith's [13] series included 56.9 per cent females, Haag's [7] 61 per cent and Carvill's [2] 48.8 per cent, including patients with the congestive and those with the noncongestive type. However, if all the groups in Carvill's series are considered, women outnumbered men in the group with congestive glaucoma, comprising 57 per cent of the patients, whereas 54 per cent of the patients with noncongestive glaucoma were men. Females comprised 60.7 per cent of Weinstein's [8] series of patients, most of whom had the congestive type of glaucoma, although his smaller series of patients with simple glaucoma included only 43.8 per cent females. In Pillat's [9] Chinese series, the females outnumbered the males 3 to 1. Nelander [1] has reported twice as many females as males in both cases of the simple type and those of the congestive type. In Trantas's [10] series, males slightly outnumbered females. Rohner [11] stated a predominance of female over male patients of 3 to 2;

TABLE 9.—*Incidence of Glaucoma in Males and Females as Found by Various Investigators (Rohner)*

Investigator	Males	Females
Neuberger	127	155
Jacobsohn	65	80
Schmidt-Rimpler	1,240	1,545
Mikulinska	5.34	12.54
Kraus	126	110
Illig	218	188

this was especially marked in acute congestive glaucoma. She quoted the following figures, taken from reports of previous investigators.

In our cases, taken as a whole, there was a slight preponderance in favor of females, who comprised 51.5 per cent of all the patients. In the group of patients with acute congestive glaucoma, somewhat contrary to most reports, the sexes were evenly divided—80 and 81. The number of females was more marked in the group with chronic congestive glaucoma, the incidence being 133 females and 98 males. Patients with chronic simple glaucoma included 514 females and 509 males—an almost equal distribution. If the data are analyzed in terms of congestive and noncongestive glaucoma, it is found that 54.6 per cent of the patients with congestive glaucoma and 50.3 per cent of those with the noncongestive type were females. Generally speaking, these figures correspond with those found in Carvill's [2] study of a fairly equivalent group in the Massachusetts Eye and Ear Infirmary.

Heredity.—The hereditary tendency of glaucoma has been stated by various writers. Definite information on this was not obtainable in practically all our cases, and no conclusions could be drawn. One patient, who gave us her ancestral history, stated that she had 2 sisters affected with glaucoma.

Race.—No information as to the racial origin of the patients was obtained as a routine at the time of their admission to the clinic.

However, such information was available for all patients admitted for operation during the years 1931-1935, inclusive, and this series included 590 cases. It was thought that some light might be thrown on the question as to the prevalence of glaucoma in the Jewish race, and these patients were grouped as Jewish and non-Jewish; 13.2 per cent of the patients with glaucoma hospitalized during this time were of the Jewish race. This figure slightly exceeds the percentage of Jews in the general population of Philadelphia, which is 12.2 per cent. We do not feel that the opinion as to the greater prevalence of glaucoma in Jews is strengthened by this finding. Carvill[2] found that the Jewish race was somewhat less susceptible to glaucoma than any of the other races that were represented in her material.

Previous Treatment.—Table 10 shows the number of patients who had had therapeutic treatment before applying for consultation or treatment at the hospital.

TABLE 10.—*Number of Patients Previously Treated*

Type of Glaucoma	Type of Treatment		
	Operative	Medical	Nonmedical
Congestive.....................................	55	45	17
Simple..	180	76	22

Apparently a considerable number of patients with noncongestive glaucoma had had previous operative treatment. This is not surprising, in view of the fact that the Wills Hospital is often made use of by patients as a consulting agency, and many of this group came to the hospital as a last resort. Forty-nine patients had nonmedical supervision; they had generally applied to an optometrist, and many of these patients gave a history of frequent purchase of glasses over a short period.

Duration of Symptoms.—Table 11 summarizes the varying periods during which the patient had noticed symptoms, in the two diagnostic groups. Logically enough, patients with congestive glaucoma are more apt to apply for treatment early and patients with simple glaucoma, whose only symptom is visual loss, are more apt to defer opthalmologic consultation.

Refraction.—From the literature, it would appear that most authors agree that the glaucomatous eye is hyperopic. Priestley Smith's series of patients[13] showed the following states of refraction: hyperopia, 45.1 per cent; emmetropia, 40.9 per cent, and myopia, 14 per cent. Carvill's[2]

series showed: hyperopia, 64 per cent, and myopia, 29.2 per cent. This ratio is apparently more marked in congestive glaucoma than in the noncongestive type. Arnold Knapp[14] reported 32 cases of myopes with glaucoma, in all of whom the condition was the noncongestive type. In Weinstein's[8] series, 62 per cent of the patients were hyperopic and 14.6 per cent myopic. Rohner reported 67 cases of hyperopia, 9 cases of myopia and 130 cases of emmetropia. Our own results are shown in table 12.

The refraction was determined in only a comparatively small number of cases. Those patients whose refractive error exceeded 1 D. of

TABLE 11.—*Duration of Symptoms*

| | Type of Glaucoma | | | |
| | Congestive (337 Cases) | | Simple (1,003 Cases) | |
Duration	No.	Percentage	No.	Percentage
Less than 6 months	161	47.70	211	21.03
Less than 12 months	32	9.49	132	13.16
2 years	33	9.79	152	15.15
3 years	18	5.34	278	27.71
4 years	6	1.78	90	8.97
5 years	30	8.90	84	8.37
5 + years	57	16.90	56	5.58

TABLE 12.—*Refractive State*

Type of Glaucoma	Number of Cases		Percentage
Congestive (total cases known, 144)	Hyperopia (> + 1 D.)	35	24.3
	Myopia (> — 1 D.)	20	13.9
	Emmetropia (+ — 1 D.)	46	31.9
	Mixed	43	29.9
Simple (total cases known, 165)	Hyperopia	68	41.2
	Myopia	24	14.5
	Emmetropia	49	29.7
	Mixed	24	14.5

hyperopia or myopia were considered hyperopic or myopic, respectively. Patients with a refractive state falling within the range of + 1 to — 1 D. were considered emmetropic. These figures correspond in general with the consensus as to the comparative frequency of refractive errors in persons with glaucoma.

Systemic Conditions.—The general medical status of the patient is undoubtedly of the utmost importance in evaluating a case of glaucoma. Some authors maintain that glaucoma is but a symptom of a systemic disease, which may be vascular, neurovegetative or metabolic. Unfortunately, data as to the general medical condition of the patients are

14. Knapp, Arnold: Tr. Am. Ophth. Soc. **23**:64, 1925.

lacking in most of our cases. The systemic conditions for those patients for whom this information could be secured and the number of patients are listed as follows:

Congestive Glaucoma

Syphilis	10
Arteriosclerosis	13
Hypertension	55
Diabetes	14
Nephritis	5
Uremia	1
Arthritis	4

Simple Glaucoma

Syphilis	9
Arteriosclerosis	8
Hypertension	45
Diabetes	23
Nephritis	8
Arthritis	5
Focal infection	26
Eczema	1
Myxedema	1
Pernicious anemia	1

The general impression from this tabulation would be that the incidence of syphilis is not more than one would expect in a sample of population such as this. Arteriosclerosis has been diagnosed in those cases in which the patients were referred to the medical clinic, as was hypertension. The reports furnished were practically the same as would be expected for any group of patients of corresponding age; that is, there was a heavy incidence of hypertension, arteriosclerosis, cardiovascular disease and cardiorenal disease.

Presence of Cataract.—Considering the interference with nutrition of the lens that occurs in glaucomatous eyes secondary to pathologic changes in the ciliary body and the angle of the anterior chamber, one would expect a greater prevalence of opacities of the lens in glaucomatous patients. Carvill[2] reported that 18 of her 49 patients between 60 and 70 years of age with congestive glaucoma had cataract (37 per cent) and 20 of her 60 patients in this seventh decade of life with noncongestive glaucoma had cataract (33 per cent). In her total series of 262 cases, 146 eyes had senile cataract. The figures for the incidence of changes in the lens in the various age groups are listed in table 13, and for comparison a table is given showing the incidence of the opacities of the lens in normal eyes as found by two authors (taken from Berens[15]).

15. Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 714.

The figures for the development of cataract following operative procedures for the relief of glaucoma will be given in a later section.

Incidence of Seasonal Attacks of Acute Glaucoma.—It is a matter of interest, and perhaps of etiologic importance, to consider the season of the year during which attacks of acute congestive glaucoma developed in our patients. Weinstein [8] stated the belief that seasonal and climatic conditions are a factor in producing attacks of acute glaucoma, and in a series of 573 cases of this condition found the highest incidence to be during the winter months. Rohner [11] likewise found the

TABLE 13.—*Presence of Cataract*

Age Group	Congestive Glaucoma			Noncongestive Glaucoma		
	No. of Patients	No. with Opacities of the Lens	Percentage	No. of Patients	No. with Opacities of the Lens	Percentage
40-49	45	16	35.5	125	14	11.2
50-59	122	94	77.0	242	72	16.9
60-69	142	108	76.1	383	124	32.4
70-79	73	64	87.7	242	117	48.3
80-89	10	5	50.0	31	20	64.5
Total	392	287	73.2	1,023	347	33.9

TABLE 14.—*Percentage of Opacities of the Lens in Normal Eyes*

Age Group	Author	
	Anderson	Gradle
41-50	38.2	34.1
51-60	65.1	66.2
61-70	85.0	68.4
71 +	92.9	90.9

TABLE 15.—*Time of Year for Attacks of Acute Glaucoma*

Jan.	Feb.	March	April	May	June	July	Aug.	Sept.	Oct.	Nov.	Dec.
17	13	15	10	12	9	9	8	8	10	13	17

greatest incidence during the months from November to February, the maximum being in November. She expressed the opinion that pupillary enlargement associated with the reduced amount of daylight, changes in the barometric pressure and frequent infections of the upper respiratory tract are explanatory causes. Pillat [9] regarded meteorologic conditions as having a good deal to do with fluctuations of the intra-ocular tension. German investigators reported the highest incidence of attacks of acute glaucoma during the winter. Moynant, in India, found them most frequent in June, when the solar radiation is at a minimum in that zone.

Table 15 shows the comparative frequency of attacks of acute glaucoma in 141 cases in our series. The tendency for attacks of acute

glaucoma to occur during the cooler months of the year is somewhat borne out by these findings.

Stage When Patient Was Seen.—In our series of patients with congestive glaucoma, only 9 were seen early, with one eye apparently normal and beginning glaucoma in the other eye. Twenty-eight patients first appeared at the clinic with an early stage of glaucoma in both eyes. In 48 cases the condition was well advanced in one eye without involvement of the other, and in 51 patients the condition was in the late stage in one eye and still unilateral. Sixty patients were first seen with
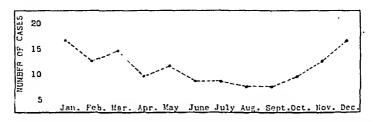
Chart 3.—Frequency of attacks of acute glaucoma in 141 cases in the months of the year. The average figures for the ten year period 1926-1935 are used.

TABLE 16.—*Stage at Which Patients with Unilateral, and Those with Bilateral, Congestive Glaucoma Were Seen*

Age Group	Unilateral (138 Cases, 41.9%)			Bilateral (196 Cases, 58.1%)			
	Early	Moderately Advanced	Late	Early	Moderately Advanced	Late	Absolute
40-49	2	10	2	8	10	6	6
50-59	3	12	13	6	22	34	8
60-69	4	18	24	8	24	38	12
70-79	..	8	11	6	3	24	18
80-89	..	..	1	..	1	6	4
Total	9	48	51	28	60	108	48
Percentage	2.6	14.5	22.4	8.3	17.8	32	14.5

both eyes involved to a well advanced degree. The largest number of patients, however, came to the clinic with late involvement of both eyes —108 persons; 48 patients had bilateral absolute glaucoma at the time of their first admission; as a group, 41.9 per cent of the patients with congestive glaucoma were first seen when the condition was unilateral, and 58.1 per cent were seen when the condition was bilateral, as shown in table 16.

Analyzing the cases of simple glaucoma in a similar way, we found that 78 patients had early unilateral simple glaucoma, 199 moderately advanced simple glaucoma and 219 late unilateral simple glaucoma. Early bilateral simple glaucoma was present in 88, moderately advanced simple glaucoma in 167 and late simple glaucoma in 227; 70 patients

did not come to the clinic until the condition had reached an absolute stage in each eye; 784 eyes had vision reduced to 6/60 when the patients first presented themselves. This comparatively large figure is a commentary on the state of knowledge of glaucoma among the general population. It is true that many of these patients came from rural districts where no facilities for diagnosis and treatment were available. As a group, 50.7 per cent of the patients with chronic simple glaucoma showed unilateral involvement, and 49.3 per cent bilateral involvement at the time of their first admission.

TABLE 17.—*Stage at Which Patients with Unilateral, and Those with Bilateral, Simple Glaucoma Were Seen*

| Age Group | Unilateral (496 Cases, 50.7%) | | | Bilateral (482 Cases, 49.3%) | | | |
	Early	Moderately Advanced	Late	Early	Moderately Advanced	Late	Absolute
20-29	..	..	..	..	..	..	..
30-39	..	..	..	..	..	..	..
40-49	23	30	35	27	44	39	6
50-59	20	58	56	25	38	59	20
60-69	22	72	59	23	56	79	21
70-79	13	36	58	12	29	45	14
80-89	..	3	11	1	..	5	9
Total........	78	199	219	88	167	227	70
Percentage of 978 cases....	7.9	20.3	21.8	8.9	17.0	23.2	

FOLLOW-UP STUDY

With the exclusion of 768 patients who resided outside of Philadelphia and with whom no attempt at contact was made, the follow-up and field aspect of the study concerns itself with 1,108 patients, residents of Philadelphia, who at various times during the period from 1926 to 1935 had appeared at the clinics of the Wills Hospital and whose condition was diagnosed as glaucomatous (including patients with secondary glaucoma).

Letters were addressed to the homes of these 1,108 patients, requesting them to appear at a stated hour at the hospital for further examination of their ocular condition; 573, or 56.8 per cent, of these letters were returned by the post-office as undeliverable. No further attempt was made to contact these persons.

It was assumed that the remaining 535 letters had been delivered and that the patients had been notified of the hospital's request. A certain number of persons began to appear for examination. After waiting a reasonable period, field workers (registered nurses) were sent to interview those patients who had not appeared and to assist them in reaching the hospital. Of this group, 290 patients were contacted in their homes by nurses; 245 persons could not be located, although in certain instances information concerning the patient was obtained from the

neighbors, i. e., data as to death, the condition of eyes and other information. Two hundred and sixty-four patients in all actually reported to the hospital and were seen.

On the basis of 535 possible contacts, this is a field contact efficiency of 49.3 per cent; on the basis of 1,108 patients to be investigated, the figure for contact efficiency is 26.2 per cent. Considering the age range of the persons concerned and the heterogeneity of the group, the number of contacts made may be considered as not unsatisfactory. Table 18 outlines these data.

It was definitely determined by field workers that 61 patients had died, 14 were being treated at other hospitals, 6 were confined to insti-

TABLE.18.—*Data for Patients Considered in This Study*

		Patients Residing in Philadelphia		
	Letters Returned by Post Office	Letters Apparently Delivered; Field Workers Sent Out (535 Cases)		Patients Actually Reporting
Year		Patients Contacted	Patients Not Contacted	
1926	40	40	11	5
1927	101	36	13	9
1928	70	25	25	9
1929	98	26	51	28
1930	41	48	25	47
1931	45	23	25	42
1932	39	21	23	24
1933	78	23	28	25
1934	42	26	29	43
1935	30	22	15	32
	573	290	245	264

Total number of patients
  Residing in Philadelphia... 1,108
  Residing outside of Philadelphia.. 768
Total number of patients reporting.. 264
  49.3% of 535 patients apparently still in Philadelphia
  26.2% or 1,108 original patients

tutions for the aged or blind, 20 were under the private care of ophthalmologists and 14 were recipients of pensions for the blind. Accordingly, the whereabouts of 115 patients was determined in addition to that of the group who reported for follow-up study.

TREATMENT

Consideration of the treatment employed in these cases is complicated by the fact that patients were under the management of nine different surgeons incumbent in the hospital at various times over the period of ten years; varying procedures were thus employed, and varying interpretations may · be placed on the results. However, in general, it may be stated that the procedure for each patient was about as follows: On the first admission to the clinic the vision was recorded with and without glasses, an external examination of the eyes and an examination of the fundi were made, and the intra-ocular tension was

measured by means of a tonometer (the Schiötz instrument was used as a routine, but there was limited use of the McLean instrument during 1926 to 1929); whenever possible, the visual fields were obtained, and medical examination was done when indicated, as were laboratory studies. The subsequent treatment was determined by the nature of the case, and no uniform statements can be made as to the routine management of the various types of glaucoma.

The treatment falls into two groups: (1) operative and (2) nonoperative, or treatment with miotics. Operative procedures were limited to the following: (1) cyclodialysis; (2) paracentesis and posterior sclerotomy, considered as temporary alleviative measures; (3) basal iridectomy; (4) the Elliot trephine operation, and (5) the modified

TABLE 19.—*Operations Performed in the Three Diagnostic Groups*

Operation	Acute Congestive	Chronic Congestive	Simple	Total
Cyclodialysis	9	10	36	55
Paracentesis	10	6	2	18
Sclerotomy	12	25	8	45
Iridectomy	95	109	148	352
Elliot trephine operation	46	64	268	378
Lagrange operation	5	20	27	52
Operations				900
Enucleation				
Acute congestive glaucoma				12
Chronic congestive glaucoma				28
Simple glaucoma				32
Total				72
Extraction of cataract				
Congestive glaucoma				26
Noncongestive glaucoma				42
Total				68

Lagrange operation. A small group of patients were also seen who received no treatment over a period of years and can thus serve, in a sense, as a control series.

Table 19 enumerates the various operations performed in the cases of the three diagnostic groups of glaucoma. In all, 900 operative procedures were performed to reduce the intra-ocular tension. In the entire series, 72 enucleations were done and 68 operations for extraction of cataract performed on glaucomatous patients.

Table 20 indicates in more detailed fashion the operative procedures and the results evaluated in terms of reduction of the intra-ocular pressure. Those cases in which an intra-ocular tension below 25 mm. of mercury was maintained during the postoperative period of observation, which ranged from two to nine years, are noted under the column "Tension Reduced." It is true that a considerable number of these patients continued to use miotics during this period, but in cases

in which no secondary operation was considered necessary the primary operative result for the purposes of this survey is evaluated as satisfactory. All the figures are given in terms of individual eyes, except in the instances in which only miotics were employed, which are considered as cases. Postoperative complications, including hemorrhage of any significant amount, infection, the development of cataract and choroidal or retinal detachment, are noted for each type of operation under each variety of glaucoma.

TABLE 20.—*Results of Operations on Eyes in the Three Diagnostic Groups Evaluated in Terms of Reduction of the Intra-Ocular Tension*

Treatment	Acute Congestive			Chronic Congestive			Simple		
	Number	Tension Reduced	Tension Not Reduced	Number	Tension Reduced	Tension Not Reduced	Number	Tension Reduced	Tension Not Reduced
Cyclodialysis..................	9	3	4	10	..	5	36	14	18
Complications (postop.)...	Hemorrhage, 2			Hemorrhage, 1			Cataract, 1; hemorrhage, 2		
Paracentesis..................	10	6	2	6	4	2	2	..	..
Sclerotomy....................	12	8	1	25	14	4	8	6	..
Complications (postop.)...	....			Hemorrhage, 1					
Iridectomy....................	95	40	15	109	52	11	148	60	39
Complications (postop.)...	Cataract, 2; choroidal detachment, 1; infection, 1; loss of vitreous, 1			Hemorrhage, 2; cataract, 2; retinal detachment, 1			Hemorrhage, 3		
Elliot trephine operation.......	46	24	6	64	29	12	278	165	59
Complications (postop.)...	Cataract, 1; choroidal detachment, 1			Cataract, 2; infection, 1			Cataract, 9; hemorrhage, 8		
Modified Lagrange operation..	5	2	3	20	6	9	27	17	7
Complications (postop.)...	....			Infection, 1					
Miotics (cases)................	23	4	1	69	6	10	257	74	78

Table 21 expresses in percentage form the tension-reducing efficiency of each operation for each type of glaucoma. In this table the operations paracentesis and sclerotomy are not considered, since it is felt that they are but temporary expedients and should not be included in the final disposition of the case.

It would seem hazardous to evaluate the worth of an operative procedure in these terms, but this has been done by various authors, a few of whom can be cited for comparison. Arnold Knapp,[16] in treating 200 patients with chronic glaucoma, obtained successful reduction of

16. Knapp, Arnold: Operative Treatment of Chronic Glaucoma: Report of Two Hundred Successful Operations, Arch. Ophth. **10**:298 (Sept.) 1933.

tension in 5 of 12 by iridectomy, in 60 of 80 by the Elliot trephine operation, in 85 of 95 by the Lagrange operation and in 5 of 8 by cyclodialysis. Etienne [17] obtained reduced tension in 114 of 187 cases; he employed the trephining operation in 89 and the Lagrange operation in 29. Kotljarevskaja obtained reduced tension in 97 per cent of cases in which the Elliot trephining operation was done. Philippow, in a series of 54 cases, after cyclodialysis obtained reduced tension in 98.1 per cent. Bothman and Blaess [18] used the Elliot trephine operation in 70 cases and obtained a reduction of tension in 98.2 per cent.

Our own series, which includes instances of all stages, would seem to indicate that the Elliot operation has comparatively the highest efficiency in simple glaucoma. As a whole, cyclodialysis does not appear to be of great value, although it must be remembered that in many of the cases in which this operation was utilized the condition was

TABLE 21.—*Tension-Reducing Efficiency of Operations for Each Type of Glaucoma*

Treatment	Acute Congestive			Chronic Congestive			Simple		
	No. of Eyes Followed	Tension Reduced	Percentage	No. of Eyes Follower	Tension Reduced	Percentage	No. of Eyes Followed	Tension Reduced	Percentage
Cyclodialysis..................	7	3	43	5	0	0	32	14	44
Iridectomy....................	55	40	73	63	52	83	99	60	61
Elliot trephine operation.......	30	24	80	41	29	71	224	165	74
Modified Lagrange operation..	5	2	40	15	6	40	24	17	71
Miotics (cases)................	5	4	80	16	6	38	152	74	49

advanced and complicated. Basal iridectomy, as generally accepted, is more efficient in the congestive types of glaucoma, particularly when employed early, before adhesion of the root of the iris. The series in which the Lagrange operation was performed is too small to permit of any conclusions. Continued treatment with miotics in chronic simple glaucoma was efficient in only 49 per cent of the cases. This is in agreement with the thought that chronic simple glaucoma is essentially a surgical disease.

ANALYSIS OF SMALLER FOLLOW-UP SERIES

In a smaller group of cases, in which the patients appeared in person for follow-up study and could be examined somewhat more carefully, it was possible to draw conclusions that are probably more authentic.

Congestive Glaucoma.—The cases of fifty eyes that were affected with congestive glaucoma in which the patients had been followed from four to nine years are analyzed as follows with respect to the operative procedures and the visual results (table 22).

17. Etienne, quoted by Bothman and Blaess.[18]
18. Bothman, L., and Blaess, N. J.: Am. J. Ophth. **19**:1072, 1936.

Cyclodialysis was performed in 3 cases as a primary operation and was effective in reducing tension in 1 case. However, the vision deteriorated, and in all the patients absolute glaucoma was the outcome. Cyclodialysis was performed in 2 cases as a secondary operation following unsuccessful iridectomy and the Lagrange operation, respectively; the tension was not reduced in either case.

Iridectomy: There were 15 cases in which iridectomy was performed as a primary operation; the tension was reduced in 10 eyes and not reduced in 5; the eventual visual result, as judged by visual acuity and the fields, was good in only 5 eyes. In 2 cases the condition ended in the absolute stage.

Elliot Trephine Operation: This procedure was performed as a primary operation on 11 eyes and was successful in reducing the tension in 8. In 6 cases the visual efficiency was maintained as a result

TABLE 22.—*Outcome of Operations in Smaller Series of One Hundred and Sixty-Five Eyes with Congestive or Noncongestive Glaucoma*

Treatment	Number	Tension Reduced	Tension Not Reduced	Operation Successful, Percentage of Eyes	Complications
Cyclodialysis	6	1	5	16	
Iridectomy	30	19	11	63	Cataract, 1
Elliot trephine operation	59	48	11	81	Cataract, 4
Lagrange operation	10	7	3	70	
Miotics	60	42	18	70	

of the operation. The Elliot trephine operation was performed on 1 eye as a secondary operation following a previous iridectomy and was not successful. Three patients showed opacities of the lens subsequent to the Elliot trephine operation.

Lagrange Operation: This procedure was performed on 5 eyes; the tension was reduced in 4, and vision was maintained in 4; the fifth eye showed low grade postoperative iridocyclitis.

Treatment with Miotics: This treatment was continued for 13 eyes (usually the mates of eyes that had been operated on). The reduced tension was maintained in 9 cases; vision was maintained in only 7 eyes.

Comment.—Two instances may be cited for the purpose of comparing operative and nonoperative treatment. In one patient an Elliot trephine operation on the right eye was successful, whereas treatment with miotics of the left eye gave a poor result. On the other hand, in another patient a Lagrange operation on the right eye and therapy with miotics for the left eye were equally effective.

As a control instance there may be cited that of a patient seen three years previously with a condition diagnosed as glaucomatous who was frightened away and neglected to obtain any treatment.

When he was called back to the hospital for the follow-up study it was found that vision had deteriorated in the affected eye from 6/21 to perception of light.

Noncongestive Glaucoma.—A second smaller group of eyes affected with chronic simple glaucoma was also available for better analysis. This group consisted of 115 eyes. When first seen, 44 of these eyes had vision reduced below 3/60. The detailed analysis with respect to the operative procedures is as follows:

Cyclodialysis (1 case): The tension was not reduced; cataract developed, which was extracted, with no improvement in vision.

Iridectomy (15 operations): The tension was reduced in 9 eyes, in which vision was well maintained. One cataract developed in this series, which was successfully removed.

Elliot Trephine Operation (47 eyes): The tension was reduced in 43 eyes and vision was maintained in 32; operation for cataract was performed on 3 eyes in this series.

Lagrange Operation (5 cases): The tension was reduced in 3 eyes; however, in only 1 of the cases was there a good visual result.

Treatment with Miotics: Forty-seven eyes were treated with miotics; the tension was reduced in 33, in 30 of which vision was maintained.

Control Group.—Ten patients reported who had had no treatment for from two to nine years. These persons had been seen only once or twice during their original period of observation at the clinic and for varying reasons had neglected to obtain any further medical supervision. Several had been frightened away when operation was originally advised; others had expressed dissatisfaction with their original treatment and had discontinued their visits to the hospital. In these 10 patients, the condition in 11 eyes deteriorated to the point of absolute glaucoma. Thus, 1 patient, who was first seen in 1927 and who received no treatment, with an initial vision of 6/21 in the right eye and perception of light in the left, when seen in 1936 had a small temporal field in the right eye and no perception of light in the left. In another patient vision had deteriorated during the period of 1930 to 1936 from 6/60 to 6/30 to no perception of light and perception of light, respectively. On the other hand, 1 person who had an original tension of 28 mm. four years previously was found to have the same tension, the same vision and the same fields on follow-up examination.

It is apparent, however, from the findings for these control patients, that untreated glaucoma usually progresses to inevitable blindness and that ophthalmologic treatment, comparatively inefficient as it may be, is of definite value.

SUMMARY AND CONCLUSIONS

The records for 1,876 patients with glaucoma seen at the Wills Hospital during the ten years from 1926 to 1935 were examined and analyzed. This represents an incidence of glaucoma of 0.78 per cent in the patients with ocular conditions.

In the group, 413 cases of secondary glaucoma were found—22 per cent of the total series of cases.

Trauma and syphilis were the two most prominent factors in the causation of secondary glaucoma.

The development of a secondary rise in tension in any given ocular lesion is of grave significance; in 34 per cent of the cases of secondary glaucoma the condition ended in serious visual impairment.

Twenty-eight cases of congenital, and 20 cases of juvenile, glaucoma are reported.

One thousand, four hundred and fifteen cases of primary glaucoma are reported, 27.7 per cent cases of the congestive type and 72.3 per cent cases of the noncongestive type. These are analyzed on the basis of age, sex, refractive error, race, previous treatment, duration of symptoms and other factors.

The average age of the males in the series was 59.9 years, and that of the females, 59.7 years.

There appears to be no evidence for the theory that the Jewish race is more susceptible to glaucoma than other races.

There is evidence for the belief that acute attacks of glaucoma tend to occur during periods of colder, more unstable weather.

The results of the follow-up study and field work carried out among the patients living in Philadelphia are reported.

The results of treatment for the various types of glaucoma are presented from the standpoint of reduction of the intra-ocular tension during the period of observation, ranging from two to ten years.

It would appear that the Elliot trephine operation is the most efficient form of operative treatment.

A small control group of patients receiving no treatment is presented to show the ultimate ocular damage in untreated patients.

From the standpoint of public health, the most significant finding is the fact that 784 eyes had vision already reduced below 6/60 when the patients first presented themselves at the hospital. This is a commentary on the state of ignorance of the early signs and symptoms of glaucoma existent among patients, to some extent among family physicians and certainly among nonmedical ocular practitioners.

ACCOMMODATION AND THE AUTONOMIC NERVOUS SYSTEM

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The rôle of the sympathetic nervous system in accommodation has been variously assumed and denied, but its active participation seems necessary to explain certain clinical and experimental phenomena to be presented in this paper. It would appear that the sympathetic system tends to adapt the eye for relatively distant objects and as such opposes the parasympathetic system, which tends to adapt the eye for relatively near objects.[1]

The mechanism whereby the sympathetic system effects this distance adjustment is not obvious. It is suggested that the radial fibers of the ciliary muscle are innervated by the sympathetic system and on contraction exert a pull on the zonule which flattens the lens. They would be opposed, of course, by the circular fibers which on contraction decrease the circumference of the ciliary ring and allow greater curvature of the lens as postulated in Fincham's[2] modification of the Helmholtz[3] theory.

Although this concept occurred to me independently it has been suggested in part by previous authors. Helmholtz[3] in 1855 considered the possibility of a dual nature of the ciliary muscle, but he discredited the idea for two reasons. In the first place, he thought the muscle fibers were too intertwined to allow of discrete actions. Second, he noted that atropine paralyzed the ciliary muscle and could do so without changing the refractive condition of the eye. As for the first objection, it is known that in other organs, such as the gastro-intestinal tract and the uterus, one finds intimate anatomic connections between antago-

1. I am aware that either a flattening or an increased curvature of the lens may be induced by factors other than distance, e. g., lenses. However, for convenience I shall use the terms "distance accommodation" and "near accommodation," as relative distance is customarily associated with the stimulus of accommodation, whatever the nature of that stimulus may be.

2. Fincham, E. F.: The Mechanism of Accommodation, Brit. J. Ophth., supp. 8, 1937, p. 5.

3. Helmholtz, H.: Ueber die Accommodation des Auges, Arch. f. Ophth. (pt. 2) **1**:1, 1855.

nistically acting groups of muscles, and apparently this does not interfere with their separate functions. As for the second objection, there is no reason to believe that atropine does paralyze the entire ciliary muscle. It is known that atropine paralyzes only such smooth muscle as is innervated by the parasympathetic system. One would not expect, therefore, the refractive condition of the eye to be changed under the influence of atropine except so far as the parasympathetic system affects it, that is, in accommodating for near and in releasing what antagonism the parasympathetic system tonically offers to the sympathetic system. The latter effect, of course, is maximal in hyperopia, in which atropinization does alter the manifest refractive condition, and minimal in myopia, in which atropinization alters the manifest condition little, if at all.

Henke [4] in 1860 postulated the theory that a ciliary muscle has two components, the circular fibers functioning for near accommodation and the longitudinal fibers for distance. Warlomont [5] (1875) made an anatomic study of the ciliary muscle and reiterated Henke's hypothesis, pointing out the fallacy of the terms "passive" and "negative" accommodation for the distance adjustment. Morat and Doyon [6] in 1891 were apparently the first to suggest that the sympathetic system effected distance adaptation and the parasympathetic system near adaptation. Henderson [7] in 1926, apparently unaware of Morat and Doyon's thesis, proposed innervation of the ciliary muscle by both the sympathetic and the parasympathetic system, stating, "Hitherto the ciliary muscle has been regarded as being actuated by one nerve only, the third nerve, in spite of the fact already noted that smooth muscle is always provided with two sets of nerves." He, like myself, was of the erroneous opinion that he was the first to postulate the existence of the dual nerve supply. He assumed that the sympathetically controlled radial fibers were "postural" and served to "fix" accommodation induced by the third nerve. This was obviously an effort to reconcile the action of the ciliary muscle with Sherrington's and Hunter's theories of the control of posture by sympathetic nerves.

Poos [8] in 1928 made the highly interesting observation that after lesions of the cervical portion of the sympathetic system epinephrine hydrochloride instilled into the eye decreased accommodation, whereas it affected normal eyes practically not at all. This is significant in

4. Henke, W.: Der Mechanismus der Accommodation für Nähe und Ferne, Arch. f. Ophth. (pt. 2) **6**:53, 1860.

5. Warlomont, E.: Le muscle ciliare, Ann. d'ocul. **73**:195, 1875.

6. Morat, J. P., and Doyon, M.: Le grand sympathique, nerf de l'accommodation pour la vision des objets éloignés, Ann. d'ocul. **106**:28, 1891.

7. Henderson, T.: The Anatomy and Physiology of Accommodation in Mammalia, Tr. Ophth. Soc. U. Kingdom **46**:300, 1926.

8. Poos, F.: Zur Frage der sympathischen Innervation des Ziliarmuskels und Ihrer Bedeutung für die Akkommodation, Klin. Monatsbl. f. Augenh. **80**:749, 1928.

view of the known sensitization of structures innervated by the sympathetic system to epinephrine after lesions of the pathways of the sympathetic system. Hudelo [9] in 1930 stated that the assumption of the existence of a ciliary muscle with innervation by antagonistic systems is necessary for an understanding of the rapidly occurring refractive changes seen in diabetes and other diseases. Luedde [10] in 1932, on the contrary, stated: "According to all available facts, it seems unlikely that the mechanical readjustments of the tissues incident to accommodation are modified by sympathetic enervation."

Byrne [11] in 1933, using an instrument which he called an oculomanometer, showed by rather elaborate experiments that stimulation of the sympathetic system induced a flattening of the lens and stimulation of the parasympathetic system induced an increased curvature of the lens. An oculomanometer as devised by him consists of "a balance with a long and a short arm, the former carrying the writing point, whilst the latter carries a rigid, adjustable, vertical rod which descends from, and at right angles to, the end of the short arm . . . until its free end rests upon . . . the center of the lens," a small trephine hole having been made in the cornea. He also showed that both the increase and the decrease in convexity, respectively, could be sensitized to the action of pilocarpine and epinephrine by prior section of the respective autonomic nerves. Byrne further assumed that the radial portions of the ciliary muscle are innervated by the sympathetic system and the circular portions are innervated by the parasympathetic system. On studying Byrne's report I was impressed by the identity of my conclusions with his. However, there are several potential objections to Byrne's experiments. They were performed on animals and under obviously unnatural conditions—a hole in the cornea, an evacuated anterior chamber, and a recorder on the anterior surface of the lens, which might indicate nothing more than a gross displacement of the lens. My approach, on the contrary, has been through clinical observations on the intact eye, and I have come to the same conclusions. I believe that I have substantiated by studies on man Byrne's conclusions based on experiments on animals.

Nicolai [12] in 1935 assumed a dual action of the ciliary muscle, with an antagonism between the radial and circular portions, and suggested

9. Hudelo, A.: Mécanisme de l'accommodation et myopie diabétique, Arch. d'opht. **47**:70, 1930.

10. Luedde, W. H.: The Mechanism of Accommodation, Facts and Fancies, Arch. Ophth. **7**:40 (Jan.) 1932.

11. Byrne, J. G.: Studies on the Physiology of the Eye, London, H. K. Lewis & Co., Ltd., 1933.

12. Nicolai, C.: Der Mechanismus der Akkommodation, Klin. Monatsbl. f. Augenh. **94**:617, 1935.

on a priori grounds that the two components might be innervated by the sympathetic and the parasympathetic system. He presented only hypothetic evidence to corroborate his assumption. Strebel [13] in 1936 considered a dually acting ciliary muscle, but rejected this concept, as did Helmholtz, largely on the basis of the results of atropinization (and also because of his inability to show a biphasic action current of the ciliary muscle). He stated that were there a muscular apparatus the contraction of which accommodates the eye for distance, one would have to make the unlikely assumption that it is not paralyzed by atropine but rather that atropine brings about a cramplike contraction. This is exactly what I propose. Why he believed that this is unlikely he did not state, but certainly this contractive effect of atropine has its counterpart in every other structure innervated by a dual system, e. g., the iris, in which atropinization allows a cramplike contraction of the muscle innervated by the sympathetic system.

THEORETICAL CONSIDERATIONS

a. Evidence for Dual Innervation of the Eye by Analogy.—Those internal organs the functions of which are regulated by involuntary muscle are innervated by a dual system, the two components of which are mutually antagonistic. These two components are, of course, the sympathetic and parasympathetic nervous systems. In the case of the heart, for example, impulses from the sympathetic division are cardiac accelerators, while those emanating from the parasympathetic division are cardiac depressors. The same antagonism holds for other organs. In some cases the sympathetic system is excitatory, while in other cases the parasympathetic system is excitatory. In the gastro-intestinal tract, unlike the heart, it is the parasympathetic system which excites motility, while the sympathetic system has the reverse effect. Thanks to the work of Cannon [14] and others, the dominance of the one or the other system has been shown to have biologic utility. The sympathetic syndrome is invoked in response to environmental emergencies and manifests itself in the protective reactions of fear, anger and rage. When the environment is controlled as regarding temperature and other factors, a completely sympathectomized animal may live a fairly normal life but would be unable to adjust itself to the exigencies of a variable environment. The parasympathetic system, on the other hand, functions chiefly when no emergency is imminent.

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13. Strebel, J.: Ueber die Mechanik des Akkommodationsvorganges und die Wirkungsweise der drei Fasersystem des Ciliarmuskels zur Widerlegung der Diskontinuitäts-theorien, Klin. Monatsbl. f. Augenh. **95**:235, 1935.

14. Cannon, W. B.: Bodily Changes in Pain, Hunger, Fear and Rage, New York, D. Appleton & Company, 1920.

So far, therefore, as the eye shows this antagonistic control of its autonomic functions, it is no exception, for analogous structures elsewhere in the body show a dual nerve supply.

As for the more immediate analogy between the ciliary body and the iris, it is known that accommodation is accompanied by miosis. Contraction of that portion of the ciliary muscle causing accommodation for near is accompanied by contraction of the sphincter of the iris. If the ciliary muscle is thought of as a single unit and under the influence of only one branch of the autonomic system, a similar mechanism would be expected in the iris. Such was conceived to be the case prior to the recognition of the dilator muscle by Langley and Anderson [15] in 1892.[16] Theretofore, the pupil was generally believed to be under the sole control of the sphincter, which produced miosis through contraction and mydriasis through inhibition. Whatever part inhibition of the sphincter plays, it is now known that the positive force of mydriasis is contraction of the sympathetic dilator muscle, and I believe that a similar positive force accommodating the eye for distance is present in the radial fibers. The radial fibers would seem to me to be synergistic with the dilator pupillae, just as the circular portion is synergistic with the sphincter pupillae. Mydriasis is associated with distance, just as miosis is with near accommodation.

Poos [8] emphasized the analogy between the innervation of the ciliary muscle by the sympathetic system and that of the iris on pharmacologic grounds. He noted that cocaine affects the normal iris and normal ciliary body but has practically no effect on either after section of the cervical portion of the sympathetic chain, whereas instillations of dilute epinephrine hydrochloride have practically no effect on the normal iris or ciliary body but have profound effects on both after lesions of the cervical portion of the sympathetic system.

By analogy, therefore, with other smooth muscle, and especially with that of the iris, the ciliary muscle may be expected to have a double innervation. Many of the discrepancies which formerly applied to the iris also apply to the ciliary muscle unless such a double function is assumed.

b. Anatomicophysiologic Factors.—The muscle fibers of the ciliary body are generally divided into three groups according to their direction: (1) circular fibers, which are concentric with the lens, forming a con-

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15. Langley, J. H., and Anderson, H. K.: On the Mechanism of the Movements of the Iris, J. Physiol. **13**:554, 1892.

16. It is of interest to note that one of Langley and Anderson's objections to the alleged control of the pupil purely by the parasympathetic system was the partial miosis in Horner's syndrome. One of my major objections to considering the ciliary muscle as controlled purely by the parasympathetic system is, similarly, the otherwise inexplicable accommodative phenomena associated with Horner's syndrome.

tinuous and endless muscle belly, sphincter-fashion; (2) radial fibers, originating at the immobile scleral spur and radiating fanwise toward the surface of the ciliary body, and (3) meridional fibers, which also originate at the scleral spur and which extend toward the choroid. The groups, especially the circular and radial, are closely intertwined, and unless one has a meridional cross-section of the eye it may be impossible to differentiate the fibers.

So much for the ciliary muscle. The zonular fibers, through which the muscle acts, arise over the entire surface of the ciliary body, except for the apexes of the processes, and are attached chiefly to the para-equatorial portions of the lens. There is some dispute as to the origin of the individual fibers, but evidence to date would seem to be against the earlier impression that the fibers attach directly to the epithelium of the ciliary body. Wolfrum [17] has traced the fibers through the epithelial layer into the subepithelial tissue. As the fibers approach the lens they are arranged in a concentric fashion on the anterior and posterior capsules, each fiber having its insertion by innumerable filaments which are spread over a wide base. The anterior capsule receives about three fifths of all the zonular fibers.

On an anatomic basis, it is apparent that the sphincter-like circular fibers will tend, on contraction, to release the tension on the zonule and allow the lens to increase its curvature. The action of the radial fibers, however, is not so simple, as these fibers form an obtuse angle with the zonule. Their action would be twofold, a forward and an outward pull on the ciliary end of the zonule, the former effect being maximal on the posterior muscle fibers and the latter pull maximal on the anterior fibers. It will be remembered that the bulk of the radial fibers are placed anteriorly. The anterior displacement would in itself tend to release the tension on the zonule were it not compensated and, in my opinion overbalanced by the outward movement which increases the zonular tension and flattens the lens. This anterior displacement may have the effect of decreasing the angle between the radial fibers and the zonular fibers and thereby rendering the resultant outward force more nearly tangential to the anterior surface of the lens. The anterior surface is, of course, the place where the main accommodative changes take place. It appears to me, therefore, that the radial muscle fibers comprise that portion of the ciliary muscle which accommodates the lens for distance. At least positive distance accommodation must be accounted for in some fashion even if the specific mechanism here suggested does not meet with the reader's full agreement.

17. Wolfrum, M.: Ueber den Ursprung und Ansatz der Zonulafasern im menschlichen Auge, Arch. f. Ophth. 69:145, 1908.

I am not yet in a position to account for the function of the meridional fibers. It may be that they take no part in the accommodative act but rather function in the control of the intra-ocular tension through their action on the choroidal and ciliary vessels, similar to the action of smooth muscle on erectile tissue elsewhere. Nor am I attempting any detailed explanation of the function of the most posterior zonular fibers, which, running from the posterior surface of the lens to the posterior portions of the ciliary body, are connected with the vitreous by many fine fibrillae. It is my opinion at present, however, that these are more concerned with delimiting the vitreous than with the function of accommodation.

Unfortunately, an unwarranted terminology has been applied to accommodation which confuses the issue. Thus one speaks of accommodation as a property of changing one's focus from a distant object to a nearer object and calls the reverse relaxation of accommodation. If, as I believe, the radial fibers adapt one's focus for relatively distant objects and the circular fibers adapt one's focus for near objects, the one is just as much an accommodative function as the other. They exist in a state of reciprocity similar to that of the dilator and sphincter muscles of the pupil, and it would be simpler if there were names for them similar to mydriasis and miosis, but since such names are lacking I am referring to the two as accommodation for distance and accommodation for near, respectively.

On physiologic grounds, the relatively greater speed with which one may accommodate for distance than for near would seem to indicate that accommodation is not purely a function of the parasympathetic system. It is generally believed that smooth muscle of the autonomic nervous system is activated by some intermediary chemical, which in the case of the sympathetic system is sympathin and in the case of the parasympathetic system is acetylcholine. Were the ciliary muscle under the sole influence of the parasympathetic system, one would expect a latent period in the relaxation of the muscle for focusing on distant objects while acetylcholine was being decomposed. Although acetylcholine is very unstable, its decomposition must be slower than its generation, else no free acetylcholine would ever be present. This would mean that near accommodation would always be more rapid than distance accommodation. Subjectively, some persons find the reverse to be the case, distance accommodation being more rapid than near accommodation. Robertson,[18] using the tachistoscope of Ferree and Rand, found that those having a more rapid distance accommodation were in the minority, but the fact that any reliable group of observers have a

18. Robertson, C. J.: Measurement of Speed of Adjustment of Eye to Near and Far Vision, Arch. Ophth. **14**:82 (July) 1935.

more rapid distance accommodation than others is evidence that the phenomenon is not explicable on the basis solely of the generation of the drug of the parasympathetic system, acetylcholine.

c. Evidence for Dual Innervation from the Point of View of Comparative Anatomy.—Herbivora have practically a negligible accommodation for near, carnivora have only a moderately well developed accommodative function, while it is not until one comes to the primates that accommodation exists as one knows it in man. Thus near accommodation in the horse, cow and rabbit is practically nil. In the dog and cat it is no more than from 2 to 4 D., while in apes it is from 10 to 15 D., and in man it may be considerably more.

The biologic reason for this difference in accommodative amplitude is most succinctly expressed in the following quotation from Treacher Collins: [19]

"The herbivorous terrestrial mammals, whose safety and survival depend on their swiftness of flight from danger . . . grasp their food directly with their mouths, finding that which is suitable more by smell and by the touch of their acutely sensitive snouts than by their sight. Their visual organs must be adapted for . . . 'panoramic' vision."

On the other hand, "The terrestrial carnivorous animals require for their sustenance and survival, smell and sight which will enable them to track their prey and pounce accurately upon it when they can concentrate the sight of both eyes on their victims at the distance from which they spring."

Then, referring to the primates, he goes on to say, "As the food of these arboreal mammals consists of fruits and insects . . . they require . . . a high degree of acuity of vision for small objects."

With this in mind, it is interesting to compare the ciliary muscle of these three classes of animals. The shape of the ciliary body varies largely with the amplitude of accommodation. In herbivora the ciliary body is spindle shaped and almost entirely confined to fibers corresponding to the meridional and radial groups (Henderson [7]). This is not to be interpreted as a rudimentary muscle, for the muscle fibers are generally numerous and well developed. In carnivora there is an apparent division of the muscle; circular fibers are present but make up only a small portion of the muscle mass. The ciliary body, as it is known in man, is not attained until the circular portion is further developed. It then becomes triangular.

In other words, those animals the requirements of which necessitate a constant adaptation for distance and which are known to have prac-

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19. Collins, F. T.: Arboreal Life and the Evolution of the Human Eye, Philadelphia, Lea & Febiger, 1922.

tically no accommodation for near, appear to have a ciliary muscle composed exclusively of the noncircular portions. On the other hand, in those animals the requirements of which necessitate a ready change of focus for far and near, there appears the other muscle group, composed of circular fibers, in a degree proportionate to their respective accommodative amplitudes. If the ciliary muscle acts as a whole in the mechanism of accommodation for near, one would expect merely a hypertrophy of the already existing fibers rather than the appearance of an entirely new set. Or, conversely, one might expect a diminution of the ciliary muscle as a whole in those animals which have no near accommodation. The fact is that the ciliary muscle (meridional and radial fibers) may be well developed in the "nonaccommodating" herbivora.

I think it is fair to infer, therefore, that the meridional and radial muscles are associated phylogenetically with a focal adjustment for distance and the circular muscle with accommodation for near. A similar situation occurs in myopia and hyperopia in man. The myopic eye, being already in a state of adaptation for near, exerts relatively little demand on the circular fibers but exerts a normal or even greater demand on those fibers which are maintaining adaptation for distance. One therefore notes histologically a selective diminution of the circular fibers instead of a uniform ciliary atrophy.[20] The meridional and radial fibers are retained, and the ciliary body assumes somewhat the same shape seen in the lower animals (carnivora), which never did have a highly developed circular muscle. Conversely, in the hyperopic eye, which demands a greater accommodative amplitude, there develops a selective hypertrophy of circular fibers, not a generalized ciliary hypertrophy, and the shape becomes triangular in a manner unlike that in any of the lower animals.

d. Embryologic Factors.—In embryologic development one has an orderly recapitulation of the evolution of man. As accommodation for distance antedated accommodation for near by many phylogenetic eras, it is not surprising to find the noncircular portions of the ciliary muscle well developed in the human fetus before the appearance of any fibers of the circular group. The former appear well developed by the end of the fifth month, whereas circular fibers first appear in the sixth month, and the proportion of circular to noncircular fibers does not reach its final ratio until a varying time after birth.

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20. In fact, the bulk of the ciliary muscle in myopia is often stated to be actually greater than in hyperopia.

a. Neurologic Factors.—Clinical Observations: As I have previously suggested, if accommodation were purely a function of the parasympathetic system, lesions of the sympathetic system, whether paralytic or stimulatory, would have no influence on accommodation. That such is not the case is evident from the following case reports. As representative of paralytic lesions I have examined a group of patients who had been operated on by Dr. J. C. White with removal of portions of their sympathetic chain for Raynaud's disease, epilepsy, retinitis pigmentosa or some other condition. I have limited the subjects to those patients who had only one side operated on so that I might study the other for comparison. Since they all had Horner's syndrome postoperatively, I know I was dealing with a lesion of the sympathetic system, but it will be noted that Horner's syndrome was never complete. This is due to the fact that sympathectomy was done on only the lower cervical portion of the sympathetic system, and a complete Horner's syndrome is produced only after obliteration of the superior cervical ganglion or section of the posterior ganglionic fibers. Thus in the following cases it is only a sympathetic paresis and not a complete paralysis which I examined. I was not able to obtain for measurement a case in which sympathectomy was done on the superior cervical portion of the sympathetic system but I have every reason to expect that the findings in such a case would be even more marked.

CASE 1.—Agnes McG., aged 25, had a condition that had been diagnosed as Raynaud's disease. Three years previously the patient had sympathectomy done on the upper dorsal and the lower cervical portion of the sympathetic system on the right. A partial Horner's syndrome developed. The right pupil measured 2.5 mm. in diameter, and the left 5 mm. Refraction showed vision of 6/6 for the right eye with a + 0.25 D. sph. $\subset$ — 0.37 D. cyl., ax. 180 and the same vision for the left eye with a + 0.50 D. sph. $\subset$ — 0.37 D. cyl., ax. 180.

Accommodation (each figure represents the approximate mean of from ten to twenty measurements) was as follows: 9 D. for the right eye and 8.1 D. for the left eye, with direct measurement.

It was found, however, that the variation between successive observations was minimized by measuring the punctum proximum with a — 3 D. lens over each eye and allowing for this in the total accommodative power. I have called this the indirect measurement. With this measurement accommodation was as follows: from 10 to 10.5 D. for the right eye and from 8.5 to 9 D. for the left eye. In these measurements a pinhole of 2 mm. was used to eliminate any effect from the miosis alone.

When a — 5.50 D lens was placed before each eye and the eyes were examined separately, the patient observed that she could immediately overcome the lens with the right eye, but she experienced considerable difficulty in overcoming it with the left eye and succeeded in doing so only after an interval considerably longer than it took with the right eye. This significant observation was checked a number of times.

Believing that I should obtain a change in accommodation similar to the paradoxical pupillary reaction, I gave this patient 0.5 cc. of epinephrine hydrochloride subcutaneously, but obtained neither a pupillary nor an accommodative change.

CASE 2.—Daniel L., aged 19, had a condition that had been diagnosed as retinitis pigmentosa. Preoperative refraction showed vision of the right eye to be 6/12 with a —0.50 D. cyl., ax. 180 and that of the left eye to be the same with a + 0.25 D. sph. ⊃ — 0.50 D. cyl., ax. 180. The preoperative accommodation of each eye was 6.5 D. (mean value). The important point is that vision was the same for distance and for near, as far as the patient could judge

The patient had sympathectomy done on the upper dorsal and the lower cervical portion of the sympathetic system on the left. Horner's syndrome developed on the left. The pupil of the right eye measured 4 mm. in diameter, and that of the left eye measured 2.5 mm.; there were relative ptosis of 2 mm. and relative enophthalmos of 1 mm.

Postoperative refraction showed vision of the right eye to be 6/12 with a — 0.50 D. cyl., ax. 180 and that of the left eye to be the same with a + 0.75 ⊃ D. sph. — 0.75 D. cyl., ax. 180. The postoperative accommodation of the right eye was 7.0 D., and that of the left eye was 8.5 D.

When the patient was asked if there was any difference between his eyes preoperatively and postoperatively, he observed that postoperatively it was relatively difficult for him to maintain clear vision for distance with the left eye, although a distant object seemed clear when he first looked at it. This was not true for reading distance. The apparent instability for distance was more marked four days postoperatively than when the patient was reexamined three weeks later, although it was still definite then.

CASE 3.—William G., aged 16, had a condition that had been diagnosed as epilepsy. Vision was noted six months preoperatively to have been 20/15 for each eye, and on his own account the patient stated that his vision had been equal in the two eyes.

Sympathectomy was done on the upper dorsal and the lower cervical portion of the sympathetic system on the left. A true Horner's syndrome developed. The patient was examined four days postoperatively.

Vision of the right eye was 6/5 without a glass. Vision of the left eye was 6/9 without a glass, but improved to 6/5 with a — 0.75 D. sph.

Accommodation of the right eye was 12 D., and that of the left eye was 14.5 D. A 2 mm. diaphragm was used to exclude the effect of the miosis.

Subsequently the patient had sympathectomy done on the lower cervical portion of the sympathetic system on the left, and with the development of Horner's syndrome bilaterally accommodation was found to be equal in the two eyes.

CASE 4.—Elizabeth H., aged 46, had a condition that had been diagnosed as Horner's syndrome (on the right). Three weeks previously the patient had had a "cold," with an abscess of her right middle ear, and at that time a tender lump was noted on the right side of her neck. She had not noticed anisocoria until a few days previous to examination. The examination showed slight ptosis and enophthalmos on the right, with a pupillary measurement of 2.5 mm. for the right eye and 3.5 mm. for the left.

Refraction showed vision of the right eye to be 6/5 with a —0.50 D. cyl., ax. 180 and that of the left eye to be the same with a — 0.50 D. sph. ⊃ — 0.50 D. cyl., ax. 180. Accommodation of the right eye was from 3.5 to 4 D., and that of the left eye was 3 D.

With the right eye the patient overcame a — 2.00 D. sph. readily over the aforementioned correction, and vision for reading was 6/6, but with the left eye she could overcome only a — 1.50 D. sph. When a — 2.00 D. sph. was placed before each eye, vision of the left eye was 6/12 at best and showed an indisputable difference when compared with that of the right eye.

CASE 5.—Dorothy R., aged 20, when first examined had Horner's syndrome on the right, which was said to have been present since birth. She also had depigmentation of the right iris.

Refraction showed vision of the right eye to be 6/6 with a + 1.12 D. sph. ⊃ — 0.25 D. cyl., ax. 120 and that of the left eye to be the same with a + 1.00 D. sph. ⊃ — 0.25 D. cyl., ax. 180.

Accommodation, as measured with a 2 mm. diaphragm, was as follows: With the right eye the patient could overcome a — 10.5 D. sph. and had vision for reading of 6/9, although it was not stable. With the left eye she could overcome a — 9.0 D. sph., and vision for reading was 6/9, but she could not overcome an additional — 0.50 D. sph. even momentarily.

As the patient was a nurse and a fairly good observer, she was asked to note the difference between her two eyes while wearing minus lenses of different strengths. She observed that her ability to focus on a small letter (she used the 6/9 line) was less stable with her right eye than with her left eye. With the right eye the letters alternately faded and cleared, and she became fatigued more readily. This instability of focus with the right eye was present through the entire range of accommodation but was maximal when the eye was adapted for distance without any lens and at a minimum when the patient was using 5 D. of accommodation. She stated that she had been conscious of this instability of her right eye for years.

More significant still was the observation suggested by Dr. Verhoeff. When the patient was asked to fix with alternate eyes first on a near point and then at a distant one, she observed that it took a somewhat longer time for the distant object to come into focus with the right eye than with the left. When vertical diplopia was induced by a 5 prism diopter lens before one eye and the patient asked to look first at a near point and then at a distant one, she observed regularly that the distant image represented by the left eye came into focus more quickly and was maintained more efficiently than the image of the other eye.

On the other hand, when the patient was asked to note the difference in rate of accommodative change on looking from a distant point to a near object, she observed no difference either when she was using alternate eyes or when she observed the two images simultaneously with the vertical prism.

I was fortunate in obtaining the case history of one patient who presented the opposite condition, that induced by stimulation of the sympathetic chain. For this I am indebted to the observations of Dr. Verhoeff, who examined the patient a number of years ago. As evidence of the stimulation of the sympathetic chain there is the pupillary dilatation, in contrast to the miosis of Horner's syndrome.

CASE 6.—On Oct. 26, 1908, the patient was refracted and given the following prescription: a + 2.25 D. sph. ⊃ — 1.00 D. cyl., ax. 95 for the right eye (with which vision was 6/5) and a + 1.00 D. sph. ⊃ — 1.50 D. cyl., ax. 92 for the left eye (with which vision was 6/5).

On November 25, 1908, the patient returned. According to the record, the glasses were satisfactory until three or four days before, when vision of the right

eye was slightly blurred, especially on reading. During this time the patient had a slight cold, which had not developed into an actual cold but which was accompanied by chilly sensations. At the same time a tender spot was discovered on the right side, corresponding to the area of the superior cervical ganglion. The pupil of the right eye was slightly larger than that of the left eye. Each reacted to light and in accommodation, and the skin reflex was elicited ipsilaterally. Accommodation was slightly less in the right eye. The patient later accepted and was given a + 0.50 D. sph. in addition to the former correction. When reading through a pencil he required a + 0.50 D. sph. to make the letters appear equally distinct. The patient was examined later. The tender spot in the neck had disappeared, as had also the sense of blurring and the difference in accommodation, and he no longer tolerated the additional plus lens which had been given him.

It seems from the foregoing history that removal of portions of the sympathetic system results in a greater accommodative amplitude, while stimulation of the sympathetic system diminishes accommodative amplitude. In other words, stimulation of the sympathetic system tends to adapt the eye for distance and oppose the accommodation for near. These changes are not marked and might be overlooked in a routine examination, but they have been consistently present in the patients whom we have examined. It should be noted that most of the cases were those of recent postoperative Horner's syndrome, the effects of which may be lessened in the course of time with the regeneration of the preganglionic tracts. The fact that the lesions of the sympathetic chain in the neck have not resulted in more profound changes in the accommodation is in all probability due to the inherent autonomy of this function, like that of other functions under the influence of the autonomic nervous system. The nervous impulses vitally influence a function, but when deprived of the more proximal nervous connections the function can persist as an independent but less well ordered mechanism. Lesions of only one portion of the autonomic system, such as the sympathetic system, will impair the activity of this portion only. A paralytic lesion will render accommodation for distance less stable, while offering less resistance to accommodation for near. Stimulation has the opposite effect.

Experimental Observations: Before discussing the following experimental observations it may be well to recall briefly the structure of the autonomic system in relation to the eye.

The higher centers of the autonomic system are still disputable. But current belief would favor a center for sympathetic fibers in the hypothalamus (Karplus and Kreidl;[21] Beattie, Brow and Long;[22] Ran-

21. Karplus, J. P., and Kreidl, A.: Gehirn und Sympathicus: Ein Sympathicuszentrum im Zwischenhirn, Arch. f. d. ges. Physiol. **135**:401, 1910.

22. Beattie, J.; Brow, G. R., and Long, C. N. H.: Physiological and Anatomical Evidence for the Existence of Nerve Tracts Connecting Hypothalamus with Spinal Sympathetic Centres, Proc. Roy. Soc., London, s.B **106**:253, 1930.

son, Kabat and Magoun [23]). A similar center for parasympathetic fibers for the eye exists apparently in the midbrain below the aqueduct of Sylvius and perhaps in the hypothalamus as well. These two centers are fairly closely approximated, but the paths by which their impulses reach the eye are widely divergent.

The sympathetic fibers pass down the spinal cord to the inferior ciliospinal center of Budge (if this can be called a center), which is in the region of the lower two cervical and upper four thoracic segments. The sympathetic roots (white rami) leave the spinal cord in the region of the first three thoracic trunks, pass for the most part through the inferior cervical ganglion (or stellate ganglion, as it is called if fused with the first thoracic) and through the midcervical ganglion to reach the superior cervical ganglion. Here efferent neurons which have not terminated in the lower cervical ganglions undergo a synapse. It is to be noted that for each preganglionic neuron there are a varying number of postganglionic neurons. Thence the postganglionic fibers ascend to the base of the brain along the internal carotid artery. Here they form two interlacing plexuses called, respectively, the carotid plexus and the cavernous plexus. Thence sympathetic fibers enter the orbit with the blood vessels, with each of the motor nerves and particularly with the ophthalmic division of the trigeminal nerve. The latter innervates the intra-ocular structures by way of its nasociliary branch. The nasociliary nerve, which (it should be noted) has avoided the ciliary ganglion, gives off two long ciliary nerves. These are essentially sympathetic in nature.[24] These long ciliary nerves penetrate the sclera diagonally round the point of attachment of the optic nerve and pass forward in the perichoroidal space to take part in the elaborate plexus over the ciliary muscle. Thence terminal sympathetic fibers are contributed to the dilator muscle of the iris and, in all probability, to the ciliary muscle.

The parasympathetic fibers carrying impulses for near accommodation and pupilloconstriction do not have such a circuitous course. Coming from a center located in the midbrain, the impulses of the parasympathetic fibers are escorted directly into the orbit via the third nerve. They leave this nerve trunk after coming through the superior orbital fissure and enter the ciliary ganglion, where they pass through a synapse. The ciliary ganglion, except for a few sympathetic fibers, is a ganglion of the parasympathetic nervous system and, as was stated by Kuntz [25] in

23. Ranson, S. W.; Kabat, H., and Magoun, H. W.: Autonomic Responses to Electric Stimulation of Hypothalamus, Preoptic Region and Tectum, Arch. Neurol. & Psychiat. **33**:467 (March) 1935.

24. It is generally believed that a few sympathetic fibers from the carotid plexus are sent through the ciliary ganglion, but it is unlikely that they have a synapse here.

25. Kuntz, A.: The Autonomic Nervous System, Philadelphia, Lea & Febiger, 1929, pp. 317-323.

1929, constitutes a relay for peripheral functions. Its function is comparable to that which the superior cervical ganglion serves for impulses of the sympathetic fibers. After leaving the ciliary ganglion, the postganglionic parasympathetic fibers enter the eye through the short ciliary nerves. These, varying in number from twelve to twenty, form a cone about the optic nerve and proceed in the perichoroidal space to the aforementioned ciliary plexus. From the ciliary plexus are given off fibers to the iris and to the ciliary muscle.

It is apparent that after the sympathetic and parasympathetic fibers enter the eye an anatomic separation is practically impossible. As for the orbit, the only places where the paths are sufficiently divergent to allow separate excitation are, first, the long ciliary nerves or the nasociliary nerve from which they are derived and, second, the third nerve proximal to the ciliary ganglion. I exclude the significance of stimulating the ciliary ganglion itself because of the likelihood (Botar [26]) that it contains sympathetic as well as parasympathetic fibers. As for the neck excitatory or paralytic lesions here produce pure sympathetic effects as far as the eye is concerned. No parasympathetic fibers to the eye pass through the region of the neck. On the other hand, the only satisfactory place that a pure parasympathetic effect might be obtained with certainty would be in the intracranial portion of the third nerve.

With this anatomic discussion as a prelude, it is interesting to analyze certain pertinent experiments of others and a few of my own.

Most frequently mentioned is the classic work of Hensen and Voelckers,[27] published in 1868, which is said to prove that the ciliary muscle acts as a whole in accommodation. These authors observed changes in the ciliary body on stimulating the short ciliary nerves and the ciliary ganglion. The phenomenon of near accommodation was, of course, produced, as these tracts are predominately parasympathetic. The methods of these authors were necessarily gross, as retinoscopy was not known at that time. They inserted needles at right angles to and through the sclera in the regions overlying the ciliary body, the equator and the posterior pole. On stimulation of either the short ciliary nerves or the ciliary ganglion, there was no movement of the needles in the ciliary muscle or at the posterior pole, while the needle in the equator rotated in a direction indicating a forward displacement of the choroid. This displacement of the choroid was also observed directly by excising a window in the sclera and noting its gross movement. That these movements were accompanied by near accommodation was proved by putting

26. Botar, J.: Sur les particularités structurales des nerfs végétatifs de l'oeil, Bull. Assoc. anat., 1935, p. 15.

27. Hensen, V., and Voelckers, C.: Experimentaluntersuchung über den Mechanismus der Accommodation, Kiel, Schwarz, 1868.

a needle on the anterior surface of the lens and one on the posterior surface and noting their displacement on stimulation of the short ciliary nerves and the ciliary ganglion. The anterior surface was noted to move forward and the posterior surface backward.

That these observations made almost seventy years ago were accurate, there can be little doubt. But the conclusions which have been drawn from them are questionable. In the first place, the fact that one should get a forward displacement of the choroid in near accommodation does not prove that this displacement is due to the participation of the entire ciliary muscle. It is conceivable that contraction of only the circular fibers, which, it will be remembered, are situated at the anterior inner portion of the ciliary body, might alone exert a traction on the choroid in much the same way that the sphincter iridis muscle puts traction on the remote root of the iris. Furthermore, I might call attention to the probably dual nature of the short ciliary nerves and the ciliary ganglion. Though these are composed chiefly of parasympathetic elements, sympathetic fibers also are believed to be present, as was previously noted. Stimulation of one without the other would, of course, be impossible, although the parasympathetic fibers which predominate in these structures would preponderate in the functional manifestation of accommodation. I therefore go so far as to say that even though Brücke's muscle had been shown to contract on stimulation of these nerve fibers, one could not conclude that this was the case in the ordinary phenomena of near accommodation, nor could one conclude that such a contraction is the property of either branch of the autonomic system. On somewhat different grounds Barret [28] was similarly skeptical about Hensen and Voelckers' conclusions and stated, "They [the experiments] only prove that some movement took place within the eye on stimulation." Whether that movement was the selective action of Müller's muscle or of Brücke's muscle, separately or together, and, if the latter, what the significance of it was, is questionable.

By stimulation of the long ciliary nerves, however, or by stimulation of the cervical sympathetic chain in the neck, one would expect a pure sympathetic reflex. Both these structures have been stimulated. Jessop [29] in 1886 stimulated the long ciliary nerves of dogs and cats and observed the images of Purkinje and Sanson. The animals had previously had iridectomy. After rendering the animals somewhat myopic by pilocarpine he observed on stimulation a change in the direction of hyperopia. Morat and Doyon [6] in 1891 obtained similar results by stimulating the cervical portion of the sympathetic chain. By observing the size of the

28. Barret, J. W.: Do Mammals Accommodate? Ophth. Rev. **17**:255, 1898.
29. Jessop, M.: On the Anatomy, Histology and Psysiology of the Intraocular Muscles, Proc. Roy. Soc., London **40**:478, 1886; Physiology of the Intraocular Muscles, Ber. period. internat. Ophth.-Cong. **7**:188, 1888.

images of Purkinje and Sanson, they noted on stimulation of the sympathetic chain a flattening of the lens. These measurements, made by the phakoscopic method, required a high degree of accuracy and were necessarily difficult to perform. Hess and Heine [30] in 1898, using the skiascopic method in a dog, showed in effect the same thing. They stimulated the cervical portion of the sympathetic chain and found an increase in hyperopia of from 1.0 to 1.5 D. Langley and Anderson [15] while studying movements of the iris made some incidental observations on accommodation. They used the phacoscopic method and were unable to observe any change in the images on stimulating the sympathetic chain. It is to be noted, however, that their observations were made largely on morphinized dogs. And morphine stimulates the centers for near accommodation. That stimulation of the sympathetic chain does not have the usual response in the eye of a morphinized animal is apparent from their own observation expressed later in the same article; they stated that under the influence of morphine the pupil dilates only slightly and then irregularly when the sympathetic chain is stimulated. Römer and Dufour [31] in 1902 were unable to produce a deflection of the needle resting on the anterior surface of the lens or to demonstrate a movement of a needle stuck in the ciliary muscle on stimulating the cervical portion of the sympathetic chain. Hess and Heine had also been unable to demonstrate a deflection of a needle thrust into the ciliary muscle on stimulation of the sympathetic chain, although the animal's hyperopia increased, as shown by skiascopy. Trautvetter [32] in 1866 also reported failure to get a response to stimulation of the sympathetic chain, but he apparently used dead animals only.

In other words, definite evidence that stimulation of the sympathetic chain induces a relative flattening of the lens has been demonstrated by several investigators. Most of those who have failed to demonstrate it have also failed to produce an adequate stimulation of the sympathetic chain that would at least cause the pupil to dilate well.

The amazing thing to me is not that some have been unsuccessful in demonstrating the sympathetic flattening of the lens but that those who have done so should not have realized its significance. Thus, Jessop [29] assumed that the effects must be due to a "relaxation" of the ciliary muscle. Morat and Doyon,[6] believing the same thing, suggested that the influence of the sympathetic nervous system was merely inhibitory.

30. Hess, C., and Heine, L.: Arbeiten aus dem Gebiete der Accommodationslehre, Arch. f. Ophth. **46**:243, 1898.

31. Römer, P., and Dufour, O.: Experimentelle und kritische Untersuchungen zur Frage nach dem Einfluss des Nervus sympathicus auf den Accommodationsvorgang, Arch. f. Ophth. **54**:491, 1902.

32. Trautvetter, D.: Ueber den Nerv. der Accommodation, Arch. f. Ophth. (pt. 1) **12**:95, 1866.

That relaxation or inhibition is not the primary method of focusing for distance is apparent from direct observation of the capsule of the lens after discission and absorption of the cortical material. The tensile force exerted on the capsule is maximal in the so-called unaccommodated or "relaxed" state. When a person with the eye in such a state gazes at a distant object the capsule becomes relatively taut, and, conversely, it becomes folded on itself when the person attempts to accommodate for near (Graves,[33] 1926). It has been suggested that the elastic tissue inherent in the ciliary body keeps the lens in the flattened state while allowing stretching when the lens becomes accommodated for near. There are several points against this. In the first place, elastic tissue is not particularly abundant in the ciliary body; there is a moderate amount
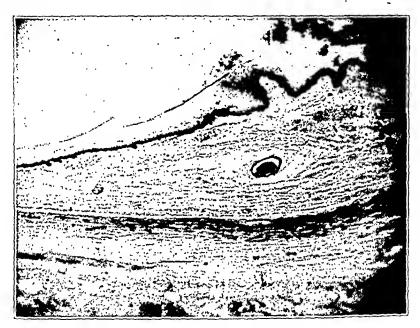
Fig. 1.—Photograph of a normal ciliary body stained with Verhoeff's elastic tissue stain, showing the paucity of elastic tissue in the ciliary body. There are practically no elastic fibers in the ciliary body except about the blood vessels, and there is a minute amount in the subepithelial tissue. As the ciliary body merges posteriorly with the more vascular choroid the elastic fibers become only slightly more numerous. In the region of the pectinate ligament there is an increase in the elastic tissue, but we are unable to agree with Fincham [2] that any one bundle would flatten the lens. The overlying sclera is richly supplied with elastic fibers and stands out by contrast in this photograph. The relative absence of elastic tissue in the ciliary body makes it necessary to postulate some force other than an elastic one to account for the phenomena of distance accommodation.

in the region of the pectinate ligament and in the sclera, but hardly any in the ciliary body itself (figure). Furthermore, a pure relationship

33. Graves, B.: Changes of Tension of the Lens Capsules During Accommodation and Under the Influence of Various Drugs, Brit. M. J. **1**:4, 1926.

between muscles and elastic tissue, in which the two components are functionally antagonistic, would be an exception to the usual architecture of the autonomic system. Of interest also is the fact that a monkey or a human being becomes accommodated for near immediately after death, that is, as soon as the sympatheticotonic state of asphyxia is passed (this is a personal observation). I consider this further evidence for the muscular control of accommodation for distance as opposed to any inherent elasticity of the ciliary body. It is my opinion, therefore, that neither accommodation for near nor accommodation for distance can be appropriately called relaxed, but that both manifestations are under the dynamic control of some portion of the ciliary muscle.

Hess and Heine,[30] after showing effectively that stimulation of the sympathetic chain caused an increase in hyperopia up to 1.5 D., suggested that with the accompanying hyperopia there was a dominance of the more peripheral lenticular refraction over the less hyperopic central portions. That this explanation was not justified is shown by my analogous experiment on a monkey and a dog, in which essentially the same procedure was used but the pupillary size was controlled by a 5 mm. aperture. I refracted the animals and then placed a plus glass before the eye, which just reversed the shadow. On stimulation of the cervical portion of the sympathetic chain there was a change in the direction of hyperopia of approximately 1 D. in the monkey and of from 0.5 to 0.75 D. in the dog. The artificial pupil did not change the findings appreciably.

A different type of experiment but one perhaps bordering on the same problem was reported by Pearcy and Allen[34] in 1927. These authors placed a balloon in the stomach and colon of their human subjects, and as the internal pressure was increased they noted a reduction of accommodation of from 2 to 5 D. Kuntz[25] in commenting on their work recalled that any somatic stimulation (e. g., pain) gives a sympathetic syndrome, as is evident in the eye from the pupillary dilatation.

In view of the foregoing clinical and experimental observations there would seem sufficient evidence to indicate that the sympathetic system had a considerable effect on the act of accommodation. Stimulation of the sympathetic chain favors a condition of adaptation for distance and antagonizes adaptation for near, while a lesion of the sympathetic system tends to impede adaptation for distance, while enhancing adaptation for near.

One may legitimately ask why adaptation for distance is at all possible when the afferent sympathetic fibers are cut and why the

34. Pearcy, J. F., and Allen, T. D.: Studies on the Visceral Nervous System: Reflexes from the Gastrointestinal Tract to the Eye, Am. J. Physiol. **82**:56, 1927.

reduction in accommodative amplitude is only from 1 to 2 D. The fact is that these sympathetic functions are capable of a great deal of autonomy, especially when the superior cervical ganglion and its post-ganglionic fibers are still intact. · Here, again, the ciliary muscle is much like the pupil, for not only are the dilator fibers still functioning many years after cervical sympathectomy, but they react even more energetically to epinephrine than does the normal pupil ("paradoxical pupillary reaction" in the sense that Duke-Elder defined it). This autonomy, as suggested by Byrne,[11] may be a reversion to a more primitive form of stimulation, that of control by hormones, or, as Cannon and Rosenbleuth[35] suggested, it may be due to increased permeability of the sympathectomized structures. In any case one cannot expect an atrophy . of the muscles supplied by the sympathetic system if they retain such a potential reaction to the proper stimulus. I was interested in examining the two eyes of a monkey which had had a unilateral sympathectomy several months previously. I was unable to demonstrate any difference in the ciliary muscles of the two eyes.

b. Pharmacologic Factors.—The autonomic nervous system is inextricably bound .up with the hormonal system. In all probability the nervous impulse results in muscular contraction only through the generation of its specific hormone. It is stated that only one in twenty of these individual muscle fibers receives a nerve ending. The others must be stimulated through a chemical or hormonal medium. The hormone for the sympathetic system is epinephrine, when produced by the adrenal glands, or its closely allied compound sympathin, when generated at the nerve ending. The injection of epinephrine hydrochloride produces in effect, then, a state of sympathetic stimulation similar to that produced by putting an electrode on the sympathetic fibers.

Accordingly, another investigator and I were given subconjunctival injections of epinephrine hydrochloride in one eye and subsequently made careful observations on the relative accommodative power of each eye, the eye into which injection had been made and the normal eye.

EXPERIMENT 1.—(D. G. C.).—Accommodation was measured a number of times before the injection; a mean value of 8 D. was obtained for each eye. Incidentally, the variability in establishing a mean was minimized by measuring accommodation with a — 3 D. lens over each eye and then finding the punctum proximum for Jaeger's test type no. 2. The 3 D. was then added to the near point measured in diopters. In this way the punctum proximum was removed to a point which allowed a greater distance per dioptric unit. All the observations were made with 2 mm. diaphragms to prevent erroneous conclusions resulting from inequality of the pupils. Two minims (0.12 cc.) of a solution of epinephrine hydrochloride was injected into the lower cul-de-sac of the left eye. In ten minutes, accommodation of the right eye was 7 D. (mean value), while accommodation of the left eye was reduced to between from 5.5 to 5.75 D. Practically the same inequality was

35. Cannon, W. B., and Rosenbleuth, A.: The Sensitization of a Sympathetic Ganglion by Preganglionic Denervation, Am. J. Physiol. **116**:408, 1936.

present in twenty minutes. In forty-five minutes accommodation was 8 D. in each eye, although the pupil was still dilated, showing again that the pupillary inequality was not responsible for the apparent difference.

The difference in the two eyes was also strikingly shown in the following manner. A — 5 D. lens was placed before each eye, and the eyes were then alternately covered. With the right eye the — 5 D. lens could be readily overcome; vision of 6/6 was obtained within a fraction of a second and maintained indefinitely. With the left eye, however, vision of 6/6 could be obtained with some difficulty and only after an interval considerably longer than with the other eye. Nor could it be maintained as stably as with the other eye, giving the impression of clearing momentarily and then fading.

EXPERIMENT 2.—(M. R. S.).—The method of examination and the results were practically identical with those in the previous experiment. The mean accommodative amplitude with the pupillary diaphragm was 8 D. for each eye. Two minims of epinephrine hydrochloride was injected subconjunctivally into the left eye. Twenty minutes later the accommodative amplitude of the right eye was between 8 and 8.25 D. and the amplitude of the left eye was 6.5 D. With a — 5 D. lens over each eye, vision of 6/6 was obtained readily and maintained with the right eye; it was obtained with greater difficulty and maintained less well with the left eye.

The action of the sympathomimetic drug epinephrine has, therefore, an effect similar to that of sympathetic stimulation in opposing accommodation for near. With the small doses used, however, we were unable to demonstrate an increase in the hyperopia. Similarly, Heath[36] in a recent communication reported a partial loss of near accommodation after instillation of the sympathomimetic drug neosynephrin hydrochloride.

Basil Graves[33] reported an interesting case, which has already been referred to, showing the effect on the capsule of the lens of cocaine, which has a sensitizing effect on the sympathetic system. The patient had apparently had a spontaneous absorption of the entire cortex and nucleus of the lens after a perforating injury. All that was left of the lens was the anterior and the posterior capsule, with a small hole in the former; the two capsular membranes were separated by aqueous. Graves observed with the slit lamp that the membranes were relatively taut when the patient focused for distance and became folded on themselves when the patient attempted to look at something near at hand. When cocaine was instilled the capsules became quite taut and ultimately showed no alteration when the patient focused for near. That this striking effect was not due to the accompanying mydriasis was shown by the fact that the buckling of the capsules on near accommodation returned before there was any diminution in the size of the pupil. If, then, as is generally believed, cocaine sensitizes the sympathetic system,

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36. Heath, P.: Neosynephrin Hydrochloride: Some Uses and Effects in Ophthalmology, Arch. Ophth. **16**:839 (Nov.) 1936.

one has a unique demonstration of this effect directly on the capsule of the lens. Graves did not, so far as I know, use epinephrine.

I have already referred to the work of Poos,[8] who showed much more striking effects from epinephrine than I did. He used instillations of epinephrine hydrochloride in patients who had had one eye sensitized by a previous unilateral lesion of the sympathetic system. This enhances the response of a structure of the sympathetic system to epinephrine. In contrast to the reduction in accommodative amplitude of 1.5 D. in our own eyes, Poos found in these sensitized eyes a reduction of 4 D. in persons having the same normal amplitude as our own.

It is interesting to review the effects of one other drug about which a good deal is known. That is atropine. Atropine is believed to "fix" or render inert the hormone acetylcholine which mediates the activity of the parasympathetic system. It thereby produces an effective paralysis of·the parasympathetic system. Such being the case, the unopposed sympathetic system might be expected to have an effect greater than usual under atropinization, and this is what one finds. Under the influence of atropine apparent hyperopia increases anywhere from 0.5 to 1 D. In myopia, on the other hand, in which there is a selective deficiency in the circular muscle, one expects the sympathetic system to be acting more nearly at its maximum effectivity all the time. One would then expect in myopia relatively little refractive change under the influence of atropine. This, of course, is what one finds; the myopic eye appears to change little, if at all, under the influence of atropine.

CLINICAL SPECULATIONS

What bearing the aforementioned concept has on clinical problems is not the primary concern of this paper. But the applications are undoubtedly manifold, and it may be well to mention a few.

Hyperthyroidism, for instance, produces a syndrome similar to that resulting from generalized stimulation of the sympathetic chain, and the ocular signs of hyperthyroidism are like those produced by stimulating the sympathetic chain. There are widening of the palpebral fissure, dilatation of the pupil and, frequently, exophthalmos, as well as an abnormal sensitivity to epinephrine (Loewi's phenomenon). I have had occasion to measure the accommodation in a number of patients during the active stages of hyperthyroidism. The results were not striking, but approximately one quarter had an accommodative amplitude less than the normal minimum for their age, Duane's [37] chart being used as the standard. I do not attach too much significance to this, as the measurement of accommodation is notably variable in any group of persons. However, it was my impression that those who had a,

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37. Duane, A.: Studies in Monocular and Binocular Accommodation with Their Clinical Applications, Am. J. Ophth. **5**:865, 1922.

diminution in their accommodation and many of the others who showed a normal range did have poorly sustained accommodation and early fatigue, compared with a normal group.[38]   It was much like the relative mydriasis which many patients with hyperthyroidism have; light causes immediate miosis but it is poorly sustained, and hippus becomes pronounced.   In any case, asthenopia is a notably frequent symptom of hyperthyroidism, varying, as Davis[39] pointed out, from "moderate discomfort to inability to use the eyes at all for near work."

Grancher[40] reported an interesting case in this connection.   His patient was a 37 year old man who could not read during his hyperthyroidism without the aid of a plus glass.   No actual measurement of his accommodation was made, but when the hyperthyroidism improved he again discarded his reading glass.

Many of the anomalies of accommodation, such as ill sustained accommodation, transient weakness of accommodation associated with migraine, and excessive accommodation, may be due in part to functional disturbances of the autonomic nervous system.   As Hudelo[9] pointed out, the alleged spasm of accommodation in asthenopes and other persons with visual disturbances is probably not a primary spasm at all but really a weakness of the muscles controlling distance adjustment. Conversely, an increased tone of muscles innervated by the sympathetic system may lead to inadequate near accommodation.   A case illustrating such a functional disturbance was brought to my attention recently by one of the Harvard University medical examiners, who stated that such cases were common in his experience.   A junior in the college came up for final semester examinations.   On the morning of the first examination he went down for breakfast and noticed that the menu was blurred. In fact, anything which he tried to read became blurred.   Somewhat alarmed, he reported to the Hygiene Building, where, aside from the blurring for near, the only findings were relatively dilated pupils, giving a startled appearance.   The pupils, however, reacted promptly to light. The boy had been well up in his studies, and he was not thought to be a malingerer.   He was, therefore, sent to the infirmary, and within a few hours, without any treatment, he was able to read without difficulty.

It may be significant that these anomalies occur in that large group of neuroses about which little is known but in which there are other evidences of disequilibrium of the autonomic system.   This would not apply, of course, to the anomalies secondary to organic changes within

38. G. Rossi (Tempi di accommodazione in ipertiroidei, Arch. di ottal. **40**:483, 1933) reported that the time for near accommodation in patients with exophthalmic goiter is retarded by from two-tenths to three-tenths second.

39. Davis, W. T.:   Ocular Symptoms of Thyroid Dysfunction, South. Med. & Surg. **95**:357, 1933.

40. Grancher: Goitre exophthalmique, Gaz. d. hôp. **53**:1060, 1880.

the eye, but it might apply to the anomalies which, usually transient in occurrence, come on, like other disturbances of the autonomic system, at times of worry or emotional distress.

SUBSEQUENT NOTE.—Since writing this paper I have come across a post-war study of Horner's syndrome in soldiers.[41] The authors reported that the near point was from 1 to 4 cm. nearer on the affected side than on the normal side. So far as I know, this is the first report of unequal accommodative amplitude in Horner's syndrome.

ABSTRACT OF DISCUSSION

DR. WILLIAM H. LUEDDE, St. Louis: Dr. Cogan found an increased capacity to accommodate, varying from 0.5 to 2.5 D., with marked contraction of the pupil on the same side, after surgical or other damage to the cervical portion of the sympathetic system in a series of five cases. Was there a control series? Was the refraction measured with the eye in complete cycloplegia? Does a 2 mm. diaphragm before the eye necessarily eliminate the effect of the inequality of the pupils?

Hess found that an apparent change of refraction of 1.5 D. or more can result solely from contraction of the pupil without any actual change in accommodation. He stated that a test with some instrument based on the principle of Scheiner is desirable to ascertain the certainty of an actual accommodative change. Were such corroborative tests made?

The history of Dr. Verhoeff's patient may be suggestive but certainly is not actual proof of innervation of the ciliary muscle by the sympathetic system. Consideration must be given to the possible influence on the accommodative capacity of the increased volume of blood in the ocular vessels after sympathectomy on the cervical portion of the sympathetic system due to paralysis or degeneration of the vasoconstrictor nerves.

The idea of an antagonistic set of muscles in the ciliary body like the dilator and sphincter fibers in the iris fails to materialize on close approach. The author has evidently overlooked Poos' statement that while he was able to produce direct contraction of isolated fibers of the dilator muscle of the iris by epinephrine, all attempts to secure similar results on preparations of muscle fiber from the ciliary body of various animals resulted in failure. That observation seems to cast much doubt on the claims for an active rôle of the sympathetic nerves in the ciliary musculature.

Stotler and Clark, working independently, reached identical results. Operative removal of the superior cervical sympathetic ganglion in the cat results in degeneration of the fibers supplying the dilator muscle of the iris but not of those supplying the sphincter muscle of the iris or any portion of the ciliary muscle. Operative removal of the ciliary ganglion in the cat results in complete degeneration of the fibers supplying the sphincter muscle or the iris and the ciliary muscle.

41. Cobb, Stanley, and Hunter, W. Scarlett: A Report of Eleven Cases of Cervical Sympathetic Nerve Injury, Causing the Oculopupillary Syndrome, Arch. Neurol. & Psychiat. **3**:636-652 (June) 1920.

Shaklee, Christensen and Kaplan found evidence of an inhibitor of sympathetic impulses on terminal fibers of the sphincter muscle of the iris but none whatever in the ciliary musculature. These reports are in full accord with the conclusions of Cannon and Rosenblueth.

The pharmacologic effects resembling those caused by stimuli of the sympathetic nerves reported by the author after injections of epinephrine hydrochloride, the effect of neosynephrin hydrochloride observed by Parker Heath and the improved cycloplegia obtained by Beach when benzedrine was combined with homatropine or atropine are probably the result of chemical inhibition in the formation or direct destruction of the acetylcholine-like substance which carries the parasympathetic impulse from the myoneural junction into the muscle cell.

What supports the proposition that so-called distance accommodation is speedier because sympathin acts on smooth muscle fibers more quickly than acetylcholine? Cannon and Rosenblueth's report on the minimal effective amounts of sympathin and acetylcholine required to evoke detectable responses on reaching a smooth muscle cell is 0.00002 Gm. for acetylcholine and three times as much, 0.00006 Gm., for sympathin.

I doubt the validity of the suggestion that primates have the greatest range of accommodation.

Any explanation for the mechanism of accommodation which fails to envisage the fact that advance of the vitreous is an inevitable sequence to contraction of the ciliary muscle is inadequate.

I would protest against the possible implications of the term gross as applied by the author to the observations of Hensen and Voelckers, made in 1867, because their work antedated the use of retinoscopy.

Donders wrote a comprehensive treatise on the "Anomalies of Refraction" without the benefit of retinoscopy.

DR. ALFRED BIELSCHOWSKY, Hanover, N. H.: Ever since I found Sherrington's law of reciprocal innervation confirmed by my own experimental and clinical investigations of the motor apparatus of the eye I have been wondering why accommodation should be the only mechanism in which two antagonistic functions would be controlled by one single nerve. In 1900, when I had discussed at the meeting of the Heidelberg Ophthalmologische Gesellschaft physiologic and clinical observations which could be explained only by the assumption of an independent mechanism for divergence, Gullstrand told me that he did not agree with my argumentation based on Sherrington's law. He stated the belief that relaxation of convergence is sufficient to produce the change from the convergent, to the parallel, and even to the divergent, position of the eyes.

It is hard to realize how the rapid and yet delicately graduated adaptation of the eye to different distances could be brought about against the resistance of elastic tissues by one nerve controlling the contraction and relaxation of the ciliary muscle.

As long ago as 1856 A. von Graefe considered it improbable that the accommodative changes are controlled by a single nerve, and W. Henke in 1860, referring to the analogy between the structure of the iris and the ciliary muscle and their intimate functional relationship, called for a correspondingly analogous innervation of the muscles which produce both pupillary and accommodative changes. A double innervation of the ciliary muscle was conceivable in view of its structure.

Since stimulation neither of the long ciliary nerves nor of the superior cervical ganglion gave unequivocal results, Poos resorted to pharmacologic stimulation of the endings of the sympathetic and parasympathetic nerves in the iris and the ciliary body. The results he obtained in normal persons when compared with those of patients suffering from lesions of the cervical sympathetic chain supported the assumption of the double innervation of the ciliary body.

Dr. Cogan's interesting observations and experiments represent an important confirmation and enlargement of Poos' findings. Poos experimented with cocaine and different epinephrine preparations. Cocaine affects the sympathetic nerves supplying the smooth muscles of the eye and its adnexa. Apart from its stimulating influence on the sympathetic nerves, cocaine is supposed to have a paralyzing effect on the parasympathetic nerves supplying the iris and the ciliary body. But this assumption cannot be reconciled with the fact that if the sympathetic fibers are paralyzed cocaine produces neither dilatation of the pupil nor paresis of accommodation, a fact suggesting that the effect of cocaine on the pupil and accommodation of normal persons may be due to stimulation of the sympathetic nerves rather than to paresis of the parasympathetic nerves. Moreover, there is a striking difference between the paresis of accommodation caused by atropine or homatropine and the paresis caused by cocaine, epinephrine or ephedrine.

Slides are demonstrated, one of which shows a graph from Poos' article, while the others illustrate the results of experiments made in our clinic. The curves show that incomplete paresis of accommodation develops as rapidly as it disappears, whereas a curve illustrating the effect of atropine shows a much slower ascent but remains for a considerable length of time at the maximum height and then returns, rather slowly, to the normal level. The difference in behavior of the accommodation just discussed is similar to the behavior of the pupil according as to whether the dilator muscle is stimulated or the sphincter paralyzed. Thus it is conceivable that cocaine and epinephrine produce not paresis of the ciliary muscle but stimulation of that portion which is controlled by the sympathetic nerves, so that while the value of the range of accommodation remains the same its position moves off with reference to the eye. If that stimulation would produce flattening of the lens one would expect a decrease of refraction. To ascertain this might be difficult, if possible at all, without the antagonistic function of the ciliary muscle being eliminated. Considering that the maximum mydriasis caused by paralysis of the sphincter muscle can be increased by cocaine, one should try to ascertain whether a decrease of refraction could be obtained by cocaine and epinephrine in suitable cases. Such a decrease would have to be attributed to flattening of the lens as a result of stimulation of the portion of the ciliary muscle controlled by the sympathetic nerves. Preliminary experiments that we have made in our institute gave negative results. After the accommodation had been completely paralyzed by atropine no decrease of the refraction could be obtained by repeated instillations of a 10 per cent solution of cocaine.

Dr. Cogan's observations on patients with an incomplete Horner's syndrome who show an increase of accommodation strongly suggest innervation of a part of the ciliary muscle by the sympathetic system.

Dr. Cogan could not ascertain a decrease of refraction in his patients or obtain it with subconjunctival injections of epinephrine in normal persons. There was just a slight diminution of accommodation according to a record concerning Dr. Verhoeff's patient with stimulation of the sympathetic fibers on one side.

Thus one may state that not only theoretical considerations but experimental and clinical observations call for innervation of the ciliary muscle by both the sympathetic and the parasympathetic system to cooperate in distance accommodation as well as in near accommodation according to Sherrington's law. For the time being, however, there is no irrefutable evidence as to flattening of the lens brought about by an independent action of the portion of the ciliary muscle controlled by the sympathetic nerves.

Dr. S. Judd Beach, Portland, Maine: As clinicians, ophthalmologists are too prone to neglect accommodation. Dr. Luedde mentioned, in connection with this subject, work by McAdams and myself with benzedrine. As was reported at the meeting of the American Ophthalmological Society, by using benzedrine with cycloplegics one avoids the nuisance of multiple instillations and at the same time speeds up recovery. When in one hour one can make one instillation of atropine and one of benzedrine do the work of nine instillations of atropine over three days the temptation is to explain this synergism according to the theory just defended by the author. While one must take care not to be seduced by this hypothesis merely because it is convenient, every evidence in favor of it is of service, and I certainly hope that Dr. Cogan will continue his investigations.

Dr. D. G. Cogan, Boston: I fear that in the few minutes I have had to think them over and in the fewer minutes that I have to discuss them I shall not be able to do justice to Dr. Luedde's criticisms, but I should like to consider as many of his comments as time permits.

First to be considered is the matter of control. I do not know what I would use as a control series. If I used normal persons with normal accommodation on the two sides I do not know which of several thousands I would choose for comparison. I should say, however, that the accommodative characteristics peculiar to Horner's syndrome are not duplicated by any constant difference in accommodation that I have seen in a series of normal persons.

Dr. Luedde suggested using a cycloplegic. Cycloplegics such as atropine and homatropine paralyze exclusively the functions of the parasympathetic system and consequently have varying effects in normal and sympathectomized eyes. In the case of the pupil, for instance, atropine produces only semimydriasis on the side on which sympathectomy was performed, in contrast to the full mydriasis on the normal side. It is my contention that comparison of these atropinized ciliary bodies would be open to the same objections as comparison of these atropinized irides; the fact is that a cycloplegic does not necessarily reduce the normal and sympathectomized eyes to a state of comparable equality, and I have purposely avoided drawing any conclusion from the findings when the eye is in cycloplegia.

Dr. Luedde suggested that the circular fibers are merely continuations of the radial group. They certainly are continuous, but this is no objec-

tion to their separate functions, as is evident in the inseparable but antagonistic muscles of the gastro-intestinal tract, or in the case of the iris, where the dilator fibers are intimately connected with the sphincter muscle.

Dr. Luedde said that the effects of the drugs reported probably indicate inhibition of the parasympathetic fibers rather than primary stimulation of the sympathetic fibers. This is a subject one might profitably dwell on at length if time permitted. The whole concept of inhibition infers that there are elastic fibers antagonizing the action of the ciliary muscle. I was rather surprised when I examined the ciliary body histologically to find a remarkable paucity of elastic fibers in this structure. This is one of the major objections to the concept. Without going into it more deeply, let me say that evidence to corroborate inhibition as the entire *vis a tergo* in distance accommodation is lacking, and until such evidence is forthcoming I do not think that one can justifiably use it to explain the action of these drugs.

I should like to emphasize again the importance of using the impartial terms near accommodation and distance accommodation rather than accommodation and relaxation of accommodation. It is not a mere quibble, for the latter terms, particularly relaxation, imply a hypothesis which it is the purpose of this report to show is inadequate. The intention of this work is to evaluate the evidence bearing on "relaxation." If the concept is found wanting, so is the name which implies it.

Let me express my sincere gratitude for Dr. Luedde's comments. Controversy makes the subject all the more interesting. I know that both Dr. Luedde and I are fascinated by this interesting subject, the last of which, I am sure, has not yet been heard. I should also like to thank Dr. Bielschowsky, with whom, of course, I am in entire accord, and also Dr. Beach for his contribution to the pharmacology of the subject.

BILATERAL ATROPHY OF THE OPTIC NERVE IN PERIARTERITIS NODOSA

A MICROSCOPIC STUDY

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AND

DAVID WEXLER, M.D.

NEW YORK

In 1929, we [1] described characteristic necrotizing lesions of the media and proliferation of the intima of the choroidal arteries in a case of periarteritis nodosa. Christeller [2] had mentioned arterial changes in the choroid, while Müller [3] had described characteristic changes in the retinal arterioles many years before. In the year our case was reported, other instances were described by Böck [4] and von Herrenschwand.[5] Böck's patient showed typical lesions in the extra-ocular muscles, and in the ciliary vessels at the entrance of the optic nerve and in their course through the sclera to the suprachoroidea. Several ocular muscles were paretic, and there was reduction in the pupillary reaction to light. The choroid was normal. In von Herrenschwand's case the lesion affected the long posterior ciliary arteries. The central retinal artery was diseased at its entrance into the optic nerve and in the region of the lamina cribrosa. The choroidal vessels were relatively uninvolved.

Helpern and Trubek [6] described lesions in the choroid in a case of gonococcic endocarditis accompanied with necrotizing arteritis which were almost identical with the choroidal lesions in our case (arteritis and proliferation of the intimal cells). This coincidence may be considered a substantiation of the view that the lesion of periarteritis nodosa is a symptom of a generalized infectious or toxic process for which no single organism can be held responsible. As far as the eye is concerned,

1. Goldstein, I., and Wexler, D.: The Ocular Pathology of Periarteritis Nodosa, Arch. Ophth. **2**:288 (Sept.) 1929.

2. Christeller: Arch. f. Verdauungskr. **37**:249, 1926.

3. Müller, in Festschrift zur Feier des fünfzigjährigen Bestehens des Stadt-Krankenhauses zu Dresden-Friedrichstadt; Dresden, W. Baensch, 1899, p. 458.

4. Böck: Ztschr. f. Augenh. **69**:225, 1929.

5. von Herrenschwand: Klin. Monatsbl. f. Augenh. **83**:419, 1929.

6. Helpern, M., and Trubek, M.: Necrotizing Arteritis and Subacute Glomerulonephritis in Gonococcic Endocarditis, Arch. Path. **15**:35 (Jan.) 1933.

Friedenwald and Rones,[7] in discussing the relation between arterial lesions in the choroid in periarteritis nodosa and those seen in septicemia, found them to be closely related, in that periarterial involvement of one kind or another was the rule. As regards the presence of so-called albuminuric retinitis, which is often seen in periarteritis nodosa when complicated by hypertensive and renal disease, they found only sclerosis of the retinal arterioles to account for the retinitis in their case. This seemed to answer the question set forth by von Herrenschwand as to whether the retinitis might be due to inflammatory lesions of the arterioles. King[8] noted papilledema resulting in atrophy of the optic nerve in a case of periarteritis nodosa, in which diagnosis was made through biopsy. In the other eye there were recurrent iritis and glaucoma which necessitated enucleation. This eye revealed retinal arteritis and periarteritis, but the lesion was not typical of periarteritis nodosa.

In Böck's[9] second case there was disease in the scleral and choroidal vessels and in the greater arterial circle of the iris. In the same article in which this case was described another case was reported, which was most interesting. In a boy of 17 progressive total detachment of the retina developed in each eye during the course of a condition proved by histologic examination to be periarteritis nodosa. With improvement in the general condition each retina reattached spontaneously, although the blood pressure had risen to 220 systolic and 165 diastolic. Several months later, vision was 20/100 in each eye. There were secondary atrophy of the optic nerve and, most striking of all, strands of pigmented tissue crossing through the fundus in its subretinal layers (retinitis striata) and apparently binding the retina to the choroid. The macula was pigmented, and below it were groups of circumscribed small masses of pigment. The arteries and veins were narrow. Under the conditions, nephritis was considered in the etiology, but Böck considered this improbable, since the patient's condition had improved, the blood urea content was not excessively high, and there was not sufficient hypertension to account for the bilateral detachment; in fact, the systolic blood pressure was only 160 and rose considerably only after reattachment. Extensive subretinal exudation as a consequence of diffuse arterial disease in the choroid was held more likely to be responsible.

The case to be reported is similar in some respects to Böck's last case, in that there were bilateral consecutive atrophy of the optic nerve and considerable pigmentary disturbance in the fundus. In addition to atrophy of the optic nerve, microscopic examination revealed extensive disease of the short posterior ciliary and choroidal arteries in each eye.

7. Friedenwald, J. S., and Rones, B.: Ocular Lesions in Septicemia, Arch. Ophth. **5:**175 (Feb.) 1931.

8. King: Tr. Ophth. Soc. U. Kingdom **55:**246, 1935.

9. Böck: Ztschr. f. Augenh. **78:**28, 1932.

REPORT OF A CASE

History.—Mrs. C. D., aged 45, had asthmatic attacks sixteen months before admission to the hospital. During the last six months she had lost much weight and suffered extreme muscular weakness. Four months previously, biopsy of a specimen of muscle at another hospital showed periarteritis nodosa. Wrist drop developed on the left. Vision suddenly became poor in both eyes. Recently there had been dyspnea, cyanosis and general parasthesias.

Physical Examination.—The patient appeared cachectic and irrational. The veins of the neck were engorged. There were several millet-sized nodules of bony consistency on the forehead. There were small nodes in the neck and others in the axillae, epitrochlear regions and groins. There were congestive signs in the lungs. The heart was enlarged, and a pericardial friction rub was heard. The liver was slightly enlarged; the spleen was not palpable. There were signs of wrist drop on the left and paralysis of the median nerve in the left hand. The blood pressure was 115 systolic and 110 diastolic. The hemoglobin content was 83 per cent. The red cells of the blood numbered 4,430,000, and the white cells 19,250, of which 91 per cent were polymorphonuclear leukocytes. The urine showed a faint trace of albumin; its concentration power was up to 1.028. There were 75 mg. of urea per hundred cubic centimeters of blood, 75 mg. of sugar and 165 mg. of cholesterol. The carbon dioxide content was 25 volumes per cent; the uric acid content was 6 mg. per hundred cubic centimeters

Externally, the eyes were clear. The pupils were semidilated and barely reacted to a stimulus of strong light. Vision of each eye was reduced to perception of movements of the hand. The fundi were similar in appearance. In the right eye the disk was pale and had a greenish tinge. There was a sharp choroidal ring in the nasal portion, but the other margins were indistinct. The surface margins of the disk were obliterated by glial tissue, only a small part of the physiologic pit being left visible. The patient was a decided brunette; however, the regular pigmentary pattern of the choroid had disappeared throughout the fundus, leaving only a diffuse fine scattering of granules of pigment. There were no clumps of pigment. The retina appeared thin, but there were no white lines or streaks. The retinal arteries and veins were narrow. The diagnosis was consecutive atrophy of each optic nerve, and it was thought that the disturbance in the pigment might be accounted for by disease in the choroidal arteries, although there was no ophthalmoscopic evidence to substantiate this belief.

Course.—The patient remained irrational. Her condition became rapidly worse. There was a preterminal rise in the temperature to 104 F. Death occurred nine days after admission to the hospital.

Autopsy.—There was periarteritis nodosa involving the vessels of the kidneys, liver, mesentery, diaphragm, spleen, lungs and heart. There were also diffuse cortical scarring of the kidneys, subcapsular atrophy and congestion of the liver, subacute suppurative pericarditis, hypertrophy and dilatation of the right auricle and ventricle and myocardial fibrosis. There were general edema, ascites, bilateral hydrothorax, and bronchial pneumonia of all the lobes of the lung.

Both globes, together with 5 mm. of the optic nerve of each eye, were removed eight hours after death. Fixation was done in Bouin's solution, pyroxylin (celloidin) and paraffin, and staining, with hematoxylin and eosin.

Microscopic Examination of the Eyes: The vessels of the iris and ciliary body were normal. The central retinal artery and the retinal arterioles, as far as they could be traced, showed no change in either eye. Changes in structure and in the vessels were confined to the episclera, the choroid and the disk in each eye.

The choroid was of normal thickness. Most of the medium-sized arteries showd infiltration of the media with mononuclear and round cells. In some there was necrosis in addition to dense infiltration; others showed proliferation of the intima, the formation of fibrin and obliteration of the lumen (fig. 1 *A* and *B*).
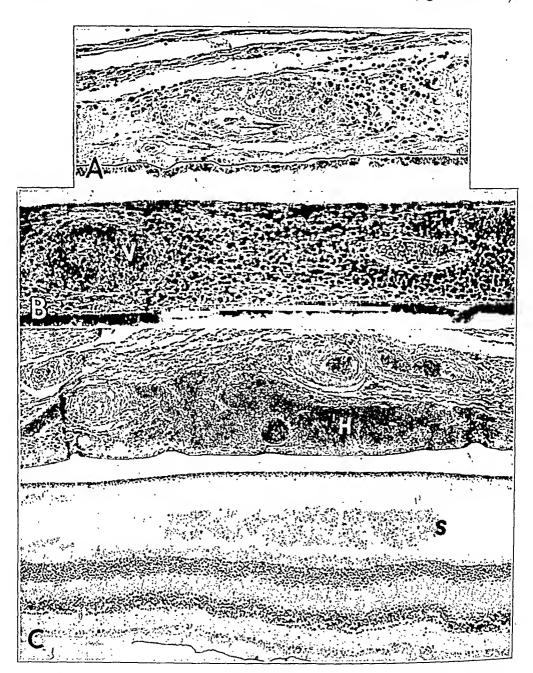
Fig. 1.—*A*, section of a medium-sized artery of the choroid, showing necrosis of the media and adventitial and perivascular infiltration. *B*, thickly infiltrated choroid; *V* indicates a necrotic infiltrated artery. The choroid is thickly infiltrated with mononuclear and plasma cells. *C*, large choroidal hemorrhage, *H*, resulting from thrombosis of a small choroidal artery; *S*, indicates subretinal exudate. The layer of rods and cones is disintegrated.
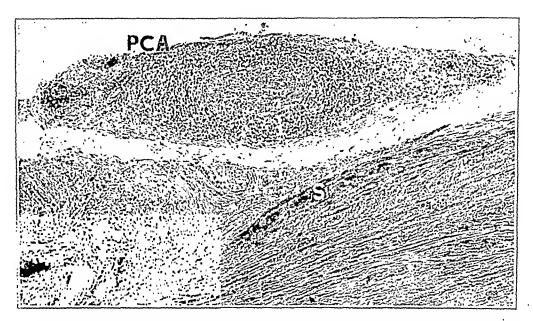
Fig. 2.—A short posterior ciliary artery, *PCA*, with highly infiltrated adventitia, media and intima; *S* indicates the sclera.

Fig. 3.—An infiltrated posterior ciliary artery, *PCA*, in longitudinal section, entering the layers of the dura, *D*, at the level of the lamina cribrosa.

Otherwise, there was moderate infiltration of the intervening structure with round cells, particularly about the blood vessels. There were a few evidences of hemorrhage in the tissue, due to rupture of the smaller vessels following fragmentation or occlusion by clots of fibrin. The capillary layer of the choroid and the elastic lamina were intact. The pigment epithelium showed little or no disturbance. The chromatophore system of the choroid was somewhat less dense than normal but showed no definite decrease in the number of cells or in their content of pigment.

The striking alterations of the retina were shrinking and fragmentation of the layer of rods and cones and its almost complete separation from the pigment

Fig. 4.—The lamina cribrosa, L, and the disk are richly infiltrated with round cells.

epithelium. In no section was there evidence of the normal continuity of these layers. Frequently there was accumulation of a granular exudate containing free pigment and pigment epithelium in the subretinal space (fig. 1 C).

In the episclera there was typical disease of the short posterior ciliary arteries, consisting of dense infiltration of all the layers, but without necrosis such as seen in the choroidal arteries. In serial section not a single vessel in this group was free from change. Similar infiltration was present in their course through the sclera and suprachoroidea. Of some interest was the disease of a vessel entering the dura and proceeding to the region of the lamina cribrosa. Such infiltration could actually be traced into the lamina and the nerve immediately in front. The optic disk contained many inflammatory round cells in addition to recently acquired glial tissue in the pit. The optic nerve was infiltrated behind the lamina and otherwise showed evidences of atrophy in its upper portion.

COMMENT

The lesions in this case correspond to those described in a great majority of the cases of this condition reported. Apparently the arteries of the ciliary system are most vulnerable. In almost every instance the choroidal vessels are affected and in some the short posterior ciliary arteries as well. In cases in which the retinal artery has been found to be diseased such change is limited in the portion behind the lamina cribrosa and not in the retinal arterioles. That the changes are restricted to these vessels conforms with the general rule that periarteritis nodosa attacks only vessels of medium size or those with well defined muscular coats. Von Herrenschwand expressed an interesting opinion regarding the site of predilection of the lesion. It appeared in his case that the periarterial nodes occurred particularly where the artery altered its direction, such as at its entrance into the sclera, its course through the lamina cribrosa and in its course from the sclera toward the ciliary body.

In the present case the posterior ciliary arteries were severely diseased, while those of the choroid were moderately so. It is fair to assume that the destruction of the layer of rods and cones and the extensive subretinal exudation are to be traced to disease in the choroid. As regards the appearance of the fundus, the widespread pigmentary disturbance may be traced to the disturbance in the neuro-epithelial layer and perhaps also to the subretinal exudate. In explanation of infiltration of the disk and the lamina cribrosa, there was sufficient indication that there had been neuritis which was dependent on disease of the ciliary arteries supplying this region (the circle of Zinn). Although the widespread disturbance of neuro-epithelium was undoubtedly a factor, it is extremely doubtful that even this could have produced such rapid loss of vision. There are, nevertheless, two distinct anatomic grounds for the loss of vision. It would appear, however, that the more rapid and greater loss is to be attributed to the local change in the nerve and that the retinal lesion was contributory. What would have ensued had the patient survived is problematic; perhaps detachment, as in Böck's last case, would have resulted, due to separation of the epithelial layer.

FITTING OF PROSTHESES FOR PATIENTS WITH CRYPTOPHTHALMOS AND EXTREME MICROPHTHALMOS

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Since ophthalmologists are beginning to realize that it is possible to fit a prosthesis in cases of congenital blindness of one eye for cosmetic improvement, many patients, varying in age from 3 months to early adult life, have been referred for such fitting. Many patients handicapped by congenital blindness have gone through life unaware that it *is* possible to fit a prosthesis without the necessity of a surgical operation—the deformity may be overcome so that it is not evident. In fact, it has been my experience over a period of many years that in such cases the natural globe presents an ideal foundation for the introduction of a prosthesis.

Usually the globe is reduced in size, but it is far superior to any implant, so that the best cosmetic results are possible. In fact, the results are vastly superior to those in cases in which an excessively large implant is used, making the proper adaptation of a prosthesis impossible. The surgeon in his endeavor to produce a perfect foundation for the prosthesis often uses an implant that is too large and does not leave sufficient room for the artificial eye. There is no advantage in using a gold or any other implant 20 or 24 mm. in diameter that results in a stump as large as the natural eye. While the stump apparently is a fine one and almost as large as the good eye, it leaves no room for the prosthesis, and the result is sure to be a failure. Besides, a large implant invariably destroys the fold of the upper lid and produces apparent ptosis.

In cases of cryptophthalmos and extreme microphthalmos the stump is small, but as the muscles are intact it is possible to fit a proper sized prosthesis, which is replaced by progressively larger ones as conditions warrant. The fear of sympathetic ophthalmia has prevented many ophthalmologists from recommending the use of a prosthesis in such cases. In my experience, no ill effects have been seen in many hundred patients who were fitted. I do not know of any case in which it was necessary to remove the globe. Of course, such patients are fitted only on the recommendation of the ophthalmologist and when his examination demonstrates that the eye is quiescent and that no danger of sympathetic ophthalmia exists.

It has been the custom of many ophthalmologists in the past to discourage or at least defer the fitting of a prosthesis for an infant. Frequently the physician advises the parent to wait "until he is 7" or until some later period or advises against the procedure. In my opinion, this is a mistake, as lack of development takes place on the affected side, and frequently permanent malformation occurs. Instead of delay, immediate action is strongly advised, so as to prevent the deformation and shrinkage that is almost certain to result. A prosthesis should be inserted even in the case of very young infants.

In many cases if shrinkage is not too great the damage may be overcome if the person doing the fitting takes the time and understands

A, appearance of the patient before the prosthesis was fitted; *B*, appearance after the prosthesis was fitted.

the proper procedure in developing the lids and bony structures by fitting progressively larger eyes as the orbit enlarges commensurate with the general body growth. Care should be exercised, and progress should be made slowly. Sufficient time should be allowed between the various fittings so as to avoid the danger of irritation to the defective globe which might cause damage to it or retard the final result.

Two cases have recently come to my attention. One child has been fitted on the advice of the ophthalmologist since the age of 3 months, and each year a larger artificial eye has been inserted. At the present time no deformity is apparent. The mother of this child met a former schoolmate who had a child of the same age with the same abnormality, which had existed for about the same time, but she had been advised

to wait "until he was older." There was marked shrinkage on the side of the face with the shrunken globe, and the lids were tight and had lost their activity through disuse. It was difficult to handle this child, as he resisted any efforts to place an eye in the socket, and it took a great deal of time and perseverance to accomplish this. After the child's confidence was gained, by periodically changing the eyes a fairly good result was obtained, but it would have been much better had the first prosthesis been fitted in early infancy. Possibly as the child develops and a larger eye is fitted each year a normal-looking socket may be obtained.

A of the figure shows the appearance of a woman 26 years of age, before the fitting of a prosthesis. She had been advised early in life that it was not possible to fit an eye and went through life thinking this to be true. When she consulted an ophthalmologist as to the condition of the eyes of her child he inquired why she did not wear an artificial eye. She informed him that her parents had been advised that it was not possible. She was referred to me, and I found that she had a perfectly movable stump. After the orbit developed as a result of fitting with progressively larger eyes for six months, an excellent cosmetic effect was obtained. The motion of the eye was good, and no deformity was apparent.

In this case there was a marked psychologic effect on the patient due to the greatly improved cosmetic effect. The importance of advising a patient with anophthalmia to resort to a prosthesis is particularly evident in this case, as the future happiness and well-being of the patient was at stake. The change of expression seen in *B* of the figure is due not to the art of photography but to the prosthesis.

Another patient with apparent anophthalmia could not secure a position as stenographer or secretary at the age of 18 owing to her appearance and the psychologic effect produced by it. The ophthalmologist consulted early in life advised that she "wait until she is older." Fortunately, little shrinkage had taken place, and in a few months the socket was normal, permitting the use of a prosthesis that matched the good eye perfectly.

There was also a marked improvement in the patient's mental attitude and her outlook on life. She promptly went out to secure a position; she got it and all the other things in life that she had denied herself owing to self-consciousness induced by her deformity. She is thankful to the ophthalmologist who advised her that she could wear an artificial eye.

AN UNCLASSIFIED TYPE OF OPTIC NEURITIS

REPORT OF CASES

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AND

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The etiology of optic neuritis covers a wide range of known possible factors, but after these factors have been carefully eliminated there remains a group of cases in which no cause can be found. These cases we classify as instances of acute infectious optic neuritis.

In the last nine months we have observed seven such cases, in which the optic disks presented a swelling varying from 1 to 6 D. In all there was sudden loss of vision with central scotomas, and in two there was no perception of light. In one case, in which the patient was first examined in 1931, we made a diagnosis of optic neuritis of unknown cause. The patient returned in February 1937, showing advanced consecutive atrophy but vision of 20/30 in each eye. Another patient, who had sudden temporary loss of vision in 1932, came to us first in March 1937. She showed atrophy of the papulomacular bundle, with vision of 20/200 in each eye. The onset in the other five patients has occurred during the past nine months. Since October 1936, our Atlanta colleagues have observed five additional cases of optic neuritis of undetermined cause. The ages of the patients varied from 5 to 22 years. In no case during the course of the neuritis were there any general symptoms, but in most instances a prodrome of malaise, slight cold and sore throat antedated the onset from one to two months. Characteristic also were soreness and pain on movement of the eyeballs. Atrophy of the optic nerve occurred early and was consecutive in type (postneuritic); its severity varied with the duration of the swelling. If vision failed to show improvement early, i. e., in six weeks, the prognosis was grave. Lillie[1] has said that in general the prognosis of optic neuritis is poor, but in his group of miscellaneous unclassified cases it was particularly so. The pallor of the optic nerve was no indication of the visual acuity.

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1. Lillie, W. I.: The Clinical Significance of Retrobulbar and Optic Neuritis, Am. J. Ophth. **17:**110-119 (Feb.) 1934.

Review of the American literature for the past decade has failed to show any specific classification of these cases. In a study of forty-three cases of true optic neuritis seen in our private practice since 1932, none was found that we could place in this group.

In these seven cases there was no history of familial disease or of sinus infection, otitis media or trauma. The Wassermann reaction of the blood of all the patients and that of the spinal fluid of the five patients whose spinal fluid was examined was negative. Roentgeno-logic studies of the cranium and sinuses, hematologic studies and urinalyses gave negative results in all the cases. The results of general physical examination and special examinations of the ear, nose and throat were also negative. Each patient was essentially emmetropic

TABLE 1.—*Age and Sex of Patients*

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Age of patient, years	16	8	17	19	19	9	12
Sex	M	M	F	M	F	F	F

TABLE 2.—*Laboratory Findings of Consequence*

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Wassermann test of the blood	0	0	0	0	0	0	0
Wassermann test of the spinal fluid	0	0	0	0	—	0	—
Other tests of the spinal fluid	0	0	0	0	—	0	—
Roentgenogram of the skull	0	0	0	0	0	0	0
Roentgenogram of the sinuses	0	0	0	0	0	0	0
Red cells	0	0	0	0	0	0	0
White cells	0	0	0	0	0	0	0
Results of urinalysis	0	0	0	0	0	0	0

under the influence of a mydriatic. Finally each patient was examined by a competent neurologist (W. A. Smith, R. B. Wilson or E. F. Fincher), with, except for the optic neuritis, entirely negative findings.

REPORT OF CASES

CASE 1.—*History.*—W. W. B., a husky, well nourished boy, was referred to us on Sept. 16, 1936. The day after a week-end at an ocean resort about two months earlier he had complained that his eyes hurt and were watering. The next day, although a rather severe cold in the head developed, he returned to work. He was not particularly drowsy, and he had no fever, as far as he knows, no frank headache and no visual disturbance. After about two weeks the cold cleared up completely.

Five weeks before consulting us the patient had difficulty in reading because his eyes watered and the letters ran together. He continued to work for three days but felt that his vision was growing worse. He consulted an ophthalmologist, who told him that his vision was "about 10 per cent in the left eye and 20 per cent in the right." A general physical examination gave negative results. Then an otolaryngologist reported clinical evidence of sinus infection. Roentgen examina-

tion did not confirm this, and on draining the sinuses no pus was obtained. Enlarged tonsils were removed three weeks before we saw him. The boy thought he could see a little better after the operation and that he could see better with the right eye than with the left. He had had slight headaches at times since the onset of his illness.

At the age of 6 years, after a cut on his foot, he had been extremely ill for several weeks; his mother thinks that he was unconscious at times during this illness, but he had no convulsions.

Ophthalmologic Examination.—Vision of the right eye was 10/200, and that of the left was 5/200. The corneas were clear. The anterior chambers were normal. The pupils were half dilated (owing to the effect of a mydriatic). The media were clear, and the disks were oval, swollen and engorged and elevated about 2 D. There were very slight edema of the periphery and marked tortuosity of vessels. The maculae were normal, and no lesions were seen in the periphery of the fundus.
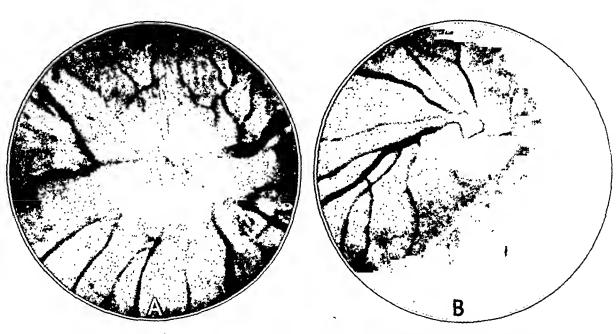
Fig. 1 (case 1).—*A,* photograph showing acute infectious optic neuritis with swelling of the disk of 2 D. *B,* photograph showing consecutive atrophy (six months after *A* was taken).

The visual fields were full peripherally but showed a central scotoma of 5 degrees in the right eye and of 10 degrees in the left.

Diagnosis.—The condition was diagnosed as acute infectious optic neuritis.

Treatment and Course.—The patient was put at absolute rest in bed. Typhoid vaccine (40,000,000 organisms) was given intravenously at intervals of five days. Fluids were limited to 1.2 liters a day, and 10 cc. of saturated solution of magnesium sulfate was given twice daily. One month later the swelling in the nerve heads had decreased to about 1 D., and the patient felt that he could see a little better. He was less nervous, and no new symptoms had appeared. Vaccine therapy was discontinued. In November the swelling in the nerve heads had practically subsided, but vision remained unchanged. He received four more injections of vaccine at five day intervals, with a considerable reaction, the temperature reaching 104 F. In January 1937 vision of the left eye had improved from 5/200 to 10/200. The right eye showed a reduction in the size of the

scotoma to 2 degrees. His general health remained good. In February there had been no great change, but the disks were fairly well outlined, and consecutive changes were beginning to appear. He was last seen in March. There had been no further improvement in vision. The atrophic change in the nerve heads was more advanced.

CASE 2.—*History.*—R. R. M., a fairly well developed, well nourished boy, was referred to us on Oct. 14, 1936. Ten days before he had complained of a headache; he had a slight cold, with disturbance in breathing at night. A few days later he experienced some pain over the left eye and on covering the right eye discovered that his left eye was totally blind. A physician was consulted immediately, but he found no abnormalities. The following day vision of the right eye began to fail, and when examined by us the patient was unable to distinguish light with either eye.

Tonsillectomy had been performed two years before, and he had had scarlet fever and pertussis in the past two years.

Ophthalmologic Examination.—The extra-ocular movements were normal in all the cardinal directions of gaze. The corneas were clear, and the pupils dilated evenly and were round. The lenses and media were clear. The nerve heads were swollen 4 D. The veins were enlarged; the arteries were somewhat tortuous, and there were a few hemorrhages over the nerve heads and some exudates adjacent to the nerve. The maculae were normal, and the remainder of the retinas showed no lesion. There was no perception of light.

Diagnosis.—The condition was diagnosed as acute infectious optic neuritis.

Treatment and Course.—The child was put to bed and given typhoid vaccine (20,000,000 organisms) intravenously; the temperature reached 103 F. This treatment was repeated every four days. Fluids were limited and saline cathartics given daily. After ten days there was some reduction in the swelling of the nerve heads. Vision had improved slightly, increasing to perception of light and color. Continuation of this regimen was urged, as the child was taken home. We have not been able to observe the boy since, but his father reports that his vision has not improved.

CASE 3.—*History.*—S. E. G., a well developed but slightly undernourished girl, was referred to us in February 1936. Four weeks previously she had begun to have burning and soreness in her lids. She had treated herself with boric acid solution but became no better. Two weeks later vision of the left eye began to blur, and the next day vision of the right eye became affected. She consulted her physician, who gave her a complete physical examination, with negative findings. Her tonsils were removed at this time "as a precautionary measure." Her vision remained blurred for the next week. Two days before we saw her she began to distinguish colors and large objects.

Her past history was essentially without significance except for frequent colds in the head during the past two years.

Ophthalmologic Examination.—Muscular movements were normal in the cardinal directions of gaze. Weakness of convergence was present. The pupils were round and dilated evenly. The lenses and media were clear. The nerve heads were swollen 1 D.; the veins were engorged, and the arteries were normal in caliber. The maculae were normal, and the retinas showed no pathologic lesion. A central scotoma of 20 degrees was present in each eye, and vision was 10/200.

Diagnosis.—The condition was diagnosed as acute infectious optic neuritis.

Treatment and Course.—The patient was put to bed and given three doses of typhoid vaccine (30,000,000 organisms) intravenously at four day intervals. She showed a marked reaction, the temperature reaching 104 F. after these injections. Fifteen days later the swelling of the papilla had receded to a barely measurable amount. There were no signs of secondary changes. She was kept in bed, however, and given two more injections of vaccine. The reaction was not so severe. She was feeling much better, and thirty days after we first saw her the disks looked fairly good; rings were visible on the temporal sides. Vision was 20/200 in each eye. She was allowed to get up and resume moderate activity. Examination in April showed only slight residual changes in the nerve head; no new symptoms had developed. Vision was 20/70 in each eye.

Comment.—The tonsillectomy was performed at an opportune time, but the patient's physician reported that the tonsils showed no pus or inspissated material.
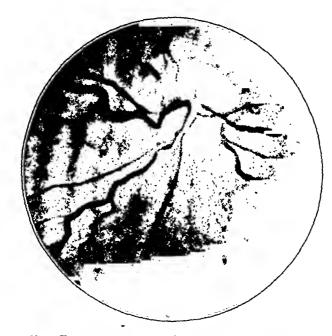
Fig. 2 (case 3).—Photograph showing acute infectious optic neuritis with swelling of the disk of 1 D.

Her sinuses were not operated on. There has been no change in her vision at this time, and the fundic picture remains the same.

CASE 4.—*History.*—R. H., a well developed, well nourished youth, was referred to us on Nov. 8, 1936. Ten days previously he had noticed dimness of vision in his left eye, which had progressed in three days to complete blindness. The next day he went blind in the right eye. He consulted his local physician, who sent him to us. The day following the onset of blindness he had pain in both eyes and rather severe headaches. Except for a mild cold in the head, he felt well.

Ophthalmologic Examination.—The ocular movements were well performed. The corneas were clear. The pupils were dilated (owing to the effect of a mydriatic). The lenses and media were clear. The disks were swollen 6 D. There were numerous flame-shaped hemorrhages over the disks, and the swelling extended somewhat into the adjacent retinas. The veins were engorged and slightly tortuous, but the arteries seemed normal. The macular areas were normal, and the peripheral part of the retinas showed no lesion.

Diagnosis.—The condition was diagnosed as acute infectious optic neuritis.

Treatment and Course.—The patient was hospitalized and given typhoid vaccine intravenously (20,000,000 organisms) every third day, the doses being increased to 170,000,000 organisms by the eighth injection. The general reaction was fairly severe, the temperature reaching 104 F. twice. After the third dose vision began to improve; objects could be distinguished, and the swelling began to subside. This improvement continued, and on December 1 vision was 20/40 in each eye. The nerve heads were swollen only 1 D.; the arteries and veins seemed normal, and the retinas and maculae were normal. The patient was dismissed from the hospital, still restricted in intake of fluids and told to resume partial activity. In December vision was 20/30 plus in each eye; the lamina cribrosa could be faintly seen, and the disks showed no beginning atrophic changes. No general symptoms have developed. In March 1937 his ophthalmologist reported that there had been no further change.

CASE 5.—*History.*—A. N. D., a well developed, well nourished young woman, consulted us in March 1937. She had applied for entrance in a nurses' training school, and routine examination showed deficient vision. There was no other complaint or symptom. In January 1932 she had been ill with influenza. A week after the onset and while she was still in bed she noticed decided blurring of vision in both eyes. This grew worse until she could barely recognize her family. This condition lasted only three days and then began to improve. She felt, however, that she had never been able to see so well after this; she always had trouble reading, although during the past two years her vision had improved.

Ophthalmologic Examination.—The ocular movements were well performed; the corneas were clear, and the pupils, which were 3 mm. in diameter, reacted to light and in accommodation. In each eye the lens and media were clear. The disk showed a decided temporal pallor, with blurred margins. The vessels were normal in size and relationship. The macula was normal, and the periphery showed no lesion. The visual fields showed central absolute scotoma of 5 degrees, with a relative scotoma of 10 degrees; examination was made with a test object of 5 degrees. Vision of each eye was 20/200.

Diagnosis.—The condition was diagnosed as consecutive optic atrophy with involvement of the papulomacular bundle. The primary cause, we feel sure, was acute infectious optic neuritis.

Comment.—This girl, of course, could not begin nurses' training. She has had no general symptoms since her illness in 1932; her health remains excellent. We believe that her condition is typical of acute infectious optic neuritis. The prognosis for any increase in her visual acuity is poor.

CASE 6.—*History.*—O. H., a well developed and well nourished girl, was seen first in April 1931. One week before, vision in her left eye had become blurred. There had been no previous serious illness. A slight cold had come on two weeks earlier. Two days before, her local ophthalmologist had found the left nerve head to be hazy and swollen and the right one normal. Vision of the right eye was 20/20, and that of the left 20/200.

Ophthalmologic Examination.—The right eye was normal. The pupil of the left eye dilated evenly and was round. The anterior chamber was normal, and the lens and media were clear. The nerve head was swollen 4 D. There was an apparent increase in the size of the capillaries over the disk. A small flame-shaped hemorrhage opposite 3 o'clock was present. The veins were tremendously engorged but showed slight tortuosity. The arteries were normal in caliber, and the macula was normal. The remainder of the retina showed no lesion.

Diagnosis.—A diagnosis of acute infectious optic neuritis was made.

Treatment and Course.—The patient was put to bed, limited in intake of fluids and given typhoid vaccine (20,000,000 organisms) intravenously at five day intervals for five doses and potassium iodide in doses of 5 grains (0.3 Gm.) daily. In May the swelling in the left nerve head had decreased to 2 D.; the hemorrhages had absorbed, and the venous enlargement was less. She was feeling physically well, but vision of the left eye remained the same. Three weeks later the swelling in the left eye was 1 D., and vision of this eye had improved to 20/70. Two days later she suddenly became blind in the right eye coincident with slight headache and malaise. There were accompanying visual hallucinations. Examination showed the right nerve head to be swollen 1 D. The swelling of the left nerve head continued to recede. She was put back to bed and given more typhoid vaccine in the vein at five day intervals. In July the swelling in the left eye had almost disappeared. There was definite consecutive atrophy present. The right eye showed a little swelling, with a return of visual acuity to 20/40. She was
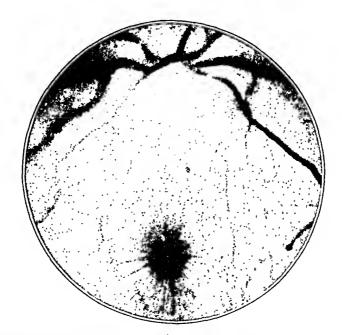
Fig. 3 (case 7).—Photograph showing acute infectious optic neuritis with a stellate figure in the macula.

allowed to get up and resume activity, as no general symptoms developed during the course of the optic neuritis. She was not seen again until October 1934. She had had no illness in this interval; she had developed normally, and her eyes had been perfectly comfortable. She had had no trouble with her school work. Each disk was distinctly pale, especially the left one. Consecutive atrophy was present, but visual acuity of each eye was 20/30. In April 1936, the findings were unchanged. Visual acuity was 20/30 in each eye; the peripheral fields were full on examination with a 5 mm. test object, and no central scotoma was elicited. Her general health had been excellent.

CASE 7.—*History.*—F. F., a well developed, well nourished girl, was examined first in December 1932. One week earlier she had noticed failing vision in her left eye. There were no other symptoms. Her general health had always been good, there having been no serious illness in her lifetime. Her eyes had never been examined, but she read a great deal without difficulty.

Ophthalmologic Examination.—The right eye was normal. In the left eye the cornea was clear; the pupil dilated evenly and was round, and lens and media were clear. The nerve head was swollen some 5 D. There were numerous flame-shaped hemorrhages over the disk; the veins were greatly engorged, but the arteries were about normal in caliber. There were a few scattered exudates adjacent to the swollen nerve. The macular region showed a clearcut star. Vision of this eye was 20/200; that of the right eye was 20/20.

Diagnosis.—A diagnosis of optic neuritis and stellate retinitis was made.

Treatment and Course.—The patient was put to bed and given intramuscular injections of 8 cc. of boiled milk at four day intervals. There was a good reaction, the temperature reaching 102 F. Two weeks later the swelling of the nerve head had decreased to 2 D.; the macular star was still present, and the vision remained the same. The patient was feeling well, and no new symptoms had developed. She was allowed out of bed. Two months later she began to menstruate, and coincident with this she felt that her vision had improved. Vision at this time was 20/100 in the left eye and 20/20 in the right. The swelling in the nerve head had decreased to 1 D. The star figure in the macula was much less marked. She was allowed to resume full activity, but was instructed to limit her intake of fluid to 1.5 liter daily and to check carefully the daily elimination. She was seen again in 1933, at which time vision of the right eye was 20/20 and that of the left eye 20/70. The swelling had disappeared. Atrophic changes were present. The macular region was clear, and the star figure had entirely disappeared. She was examined again in 1934. Vision of the left eye was 20/50; that of the right eye was normal. Her general physical condition remained good; no new symptoms had developed, and the ocular findings had not changed.

Comment.—This condition was certainly acute infectious optic neuritis with the star figure in the macula as described by Fuchs.

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GENERAL COMMENT

There is much confusion in the literature in distinguishing between optic neuritis and retrobulbar neuritis. Because of the proved central scotoma the condition in our seven cases would be classified by many as retrobulbar neuritis. In like manner, in most of the reported cases of optic neuromyelitis in which there was papillary swelling of 2 to 3 D. the condition has been diagnosed as retrobulbar neuritis. The same picture of a swollen nerve head and a central scotoma may be seen in acute syphilis of the central nervous system. We believe that retrobulbar neuritis should include only those conditions in which there is a normal fundus and that, regardless of the visual fields, every nerve head swollen because of inflammation represents optic neuritis.

It is possible that some cases of retrobulbar neuritis attributed to sinus disease in which recovery follows operation on clinically normal sinuses belong to this class. Benedict [2] expressed the opinion that foreign protein therapy would accomplish as much as surgical inter-

2. Benedict, W. L.: Retrobulbar Neuritis and Diseases of the Nasal Accessory Sinuses, Arch. Ophth. 9:893-906 (Jan.) 1937.

vention on the sinuses unless they are clinically diseased. Perhaps a similar group of cases occurs with retrobulbar neuritis as the only finding.

The fundic picture in our seven cases resembles closely that found in the demyelinating diseases associated with optic neuritis, especially neuromyelitis optica. The acute onset, the reaction and swelling of the nerve head, the central scotoma and the inevitable consecutive atrophy give a picture parallel to that of neuromyelitis optica save that the cord is not involved. It should be repeated that in the course of time the disk may take on the clearcut marginal aspect of primary atrophy: in our case the picture could easily have been mistaken for primary atrophy had the neuritic history not been known.

The sudden onset and rapid loss of vision lend weight to the theory that in these cases the condition was of infectious origin. The number of cases in this group occurring so closely together makes us feel too that the disease has epidemic characteristics. Wilson [3] stated that he has seen more cases of acute myelitis with recovery in the last nine months than in the previous seven years. Is it not possible that the same causative agent is present in these cases but with a different specificity?

Axial neuritis extending into the lamina cribrosa certainly must occur, the involvement being confined to the optic nerve, as there is no clinical evidence of involvement of contiguous tissue. The virus (if it be one) that is responsible could easily and most likely does belong in the class of large viruses or micromicrobes.[4] There is a growing opinion that multiple sclerosis is not caused by a neurotrophic virus and that the so-called cases of acute multiple sclerosis are really instances of acute encephalomyelitis or encephalitis. We realize that the condition in our cases could be mistaken for a first episode of multiple sclerosis. In multiple sclerosis, however, the early visual disturbances are usually slight and transitory, and the illness does not seem serious enough for the patient to consult a physician until general symptoms appear. The first evidence of visual disturbance in a proved case of multiple sclerosis is usually elicited in the history, and the eye is objectively normal at the time of examination. In the experience of Rivers and his collaborators,[5] there has been no proof of demyelinization being produced by any known neurotrophic viruses. Demyelinization prob-

3. Wilson, R. B.: Personal communication to the authors.

4. Rivers, T. M.: Recent Advances in the Study of Virus and Viral Diseases, J. A. M. A. **107**:206-210 (July 18) 1936.

5. Rivers, T. M.; Sprunt, D. H., and Berry, G. P.: Observation on Attempts to Produce Acute Disseminated Encephalomyelitis in Monkeys, J. Exper. Med. **58**:39-53 (July) 1933.

ably did not occur in our cases, but, unfortunately, we have no proof of the true pathologic change.

The treatment of the patients of our series has been similar; it was, in general, empirical, as we were dealing with an unknown cause. Foreign protein in varying amounts was used, care being taken to produce a moderate systemic reaction with elevation of temperature to from 102 to 104 F. The response seemed somewhat in proportion to the severity of the reaction produced. We advise complete rest and quiet for at least two weeks in all cases of this condition and longer, if necessary, depending on the response to treatment.

In one patient (case 7) who showed a stellate figure in the macula along with the neuritis the condition possibly offers a clue to the etiology of Fuchs' idiopathic retinitis.

CONCLUSIONS

We feel that the condition in these seven cases warrants a new classification, namely, acute infectious optic neuritis, the cause being an unknown virus with predilection for the optic nerve.

ABSTRACT OF DISCUSSION

DR. E. L. GOAR, Houston, Texas: The knowledge of the subject of optic neuritis is in a rather unsatisfactory state. The nomenclature, the pathogenesis, the etiology—none of these is thoroughly defined or well understood. In Duane's last translation of "Fuchs's Text-Book of Ophthalmology" (1923) papilledema is still discussed under the heading of optic neuritis. The terms retrobulbar neuritis, axial neuritis and toxic amblyopia are all used more or less synonymously. To add further to the confusion, the term interstitial neuritis has been injected, designating in this connection inflammation of the fibrous septums of the nerve, which are probably the only parts of the nerve ever primarily affected in true inflammatory lesions.

I propose that the situation be clarified by having a common understanding of the terms used, somewhat as follows: Retrobulbar neuritis should be used for a condition in which there is sudden loss of vision, with a central scotoma, but no visible pathologic changes when the eye is viewed with the ophthalmoscope. Toxic amblyopia should be used for a condition in which there is gradual loss of vision, with a central or a paracentral scotoma, which may be relative or absolute, and with no ophthalmoscopic evidence of inflammation, and which is due to some known toxin. In optic neuritis there may be any or all of the signs mentioned for the other conditions, but there must be inflammation of the disk visible with the ophthalmoscope. Papilledema, or choked disk, is a noninflammatory swelling of the disk due to increased intracranial pressure.

When one attempts to trace the etiology of the condition, the case is far more complicated. Almost every illness the human body is heir to at one time or another has had to bear the onus of optic neuritis. Syphilis, meningitis, meningo-encephalitis, sinus disease and multiple

sclerosis are all known and frequent offenders. Infections of the teeth or tonsils, diabetes, pellagra, and poisoning, such as that with lead, alcohol or nicotine, are less prominent, but they have all been indicted, along with other factors too numerous to mention. And now a filtrable virus is offered as the offending agent. I am not sure on what grounds the authors arrived at this conclusion, but certainly if all unclassified cases fall in this group, it will be fairly large. A filtrable virus has been proved to produce herpetic keratitis and has been suspected of causing sympathetic ophthalmia, and it is not beyond the bounds of reason to believe that it may attack the optic nerve.

I must say one word about sinus disease and optic neuritis. Lillie, in writing on the subject of optic neuritis a few years ago, stated that among five hundred cases of optic neuritis observed at the Mayo Clinic, in only one was the condition attributed to sinus disease. No doubt some years ago ophthalmologists were entirely too enthusiastic about sinus disease as a cause of optic neuritis, but I am convinced that the pendulum has swung too far the other way. When one sees well defined optic neuritis in a patient with purulent infection of the sphenoid sinuses or the posterior ethmoid sinuses and when the sinuses are drained and the neuritis begins to improve forthwith, that is about as good evidence of cause and effect as one is able to establish by clinical methods, even if it does savor slightly of reasoning on the principle of post hoc, ergo propter hoc. I have seen this happen in far more than 0.2 per cent of cases.

Neuromyelitis optica is a rare disease. According to Perrit's statement in 1934, only a little over fifty cases had been reported, five of them in the English literature. The disease is characterized by bilateral optic neuritis, either acute or subacute, following, as a rule, but occasionally preceded by, or simultaneous with, myelitis. As one reads the description of the disease in the reports of the cases, it seems doubtful whether this should be classed as a clinical entity or as an episode in one of the inflammatory or degenerative diseases of the central nervous system.

The cases reported by the authors resemble those classed as instances of the ocular phase of neuromyelitis optica, but the essential of the cord involvement is lacking. The solution of the problem of the etiology of what the authors have well named this condition, acute infectious optic neuritis, probably awaits the discovery of the causation of other puzzling diseases of the central nervous system, such as poliomyelitis, Parkinson's disease, multiple sclerosis, encephalitis and others, consideration of which has occupied the best minds among the neurologists for years, but which is still undetermined. Meanwhile, the virus theory offers an attractive field for speculation and experimentation.

Dr. Walter I. Lillie, Philadelphia: I had the privilege of going over Dr. Clay and Dr. Baird's paper in detail. The report of the seven cases shows that the visual loss was precipitous in onset and remained stationary for a long period. After therapy was instituted, gradual improvement was noticeable. To me, this verifies the opinion that the prognosis for optic neuritis is much more grave than that for retrobulbar neuritis. Usually the patients with retrobulbar neuritis will get well as one is sending them around to the various physicians to obtain an opinion as to what the cause might be. Optic neuritis

persists, owing, I think, to the marked swelling of the fibers of the optic nerve in the lamina cribrosa impinging against the more or less inelastic sclera, so that even a small amount of inflammation may produce tremendous damage.

The etiology in these seven cases is a moot question, because the patients fall in the age group of those with multiple sclerosis.

My associates and I had the same experience with cases that later proved to be instances of multiple sclerosis, although the first examination and subsequent examinations gave negative results after the original episode of optic neuritis.

It was also true in my experience that cases of this type after a number of years did not turn out to be instances of multiple sclerosis; so there is a small group in which the etiology cannot be definitely established.

This type of optic neuritis may occur at any age. It occurs most frequently in the age group given, but at the present time I have a patient, a man aged 74, who is in the hospital with bilateral acute optic neuritis. The nerve heads are elevated 2 D., and the patient is totally blind. He had no neurologic changes whatever, but for the past three days bulbar palsy has been present. Whether this patient belongs to the group with acute optic myelitis I do not know, but the condition might readily come under this classification.

The condition in the seventh case of Dr. Clay and Dr. Baird is very interesting because it has been given a separate name by Fuchs, stellate retinitis associated with optic neuritis. I saw a boy aged 14 who had sudden loss of central vision in each eye. This had been present about four weeks before I saw him. The slide shows swelling of the nerve heads with an incomplete macular star in each. To me, this means that this patient has had previous edema in the macular region, which is now undergoing absorption. In other words, it is a good criterion, I think, from a prognostic standpoint that the condition is getting better, and it needs no separate classification as stellate retinitis.

Dr. J. M. Baird, Atlanta, Ga.: I should like to thank Dr. Goar and Dr. Lillie for their kind remarks in discussing this paper. We realize that this subject is very controversial, but we felt, after making an exhaustive study of each of the patients and obtaining no positive findings, that this was probably the best classification we could think of for a diagnostic or disease entity.

HUMAN FACTOR IN AIRPLANE CRASHES

C. E. FERREE, Ph.D.

AND

G. RAND, Ph.D.

BALTIMORE

There seems to be a growing conviction that the pilot is an important factor in the increasing number of airplane crashes.[1] There is perhaps a good reason for this conviction. It may be that with the rapid improvement in the facilities for aviation the attitude of the pilot has changed with respect to the importance of his own qualifications, fitness and training and to the highly specialized nature of the services he was formerly called on to contribute and still has to contribute in emergencies. It may be also that not enough attention is paid to fitness in the selection of pilots and to making sure that they are in fit condition for service at all times when they are called on to render service. It seems strange that the plane should be carefully tested on every point of its construction and operation before each flight and little or no attention given to the pilot at that important time other than to see that he is on hand to fly. With the advance in scientific and mechanized control the importance of the human factor has without question been thrust into the background.

During the World War we devised an instrument for testing the fitness of aviators. This instrument was used at Mineola, N. Y., for testing and studying the fitness of candidates for entrance into the air service and later taken to France by Dr. William Holland Wilmer, surgeon in charge of the Medical Research Laboratories, Air Service, American Expeditionary Force, for the purpose of studying the fitness of aviators already in the service for the various tasks required of them, particularly in combat flying. Since the war the instrument has been

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1. Major-General James E. Fechet (Ret.), former chief of the United States Army Air Corps, for example, who has devoted a great deal of time to the study of airplane crashes, stated (Flight Surgeon Topics, School of Aviation Medicine, Randolph Field, Texas, April 1937, vol. 1, p. 44) that in more than half the number of cases these crashes are due to an error on the part of the personnel or to undetermined causes. In the personnel group he includes the following: the pilot, the weather man, the manager of the airline operations and the mechanic. Of these, the pilot is of course an important factor. A small percentage of these crashes, less than five, he said, are due to mechanical failures—malfunction of an engine or breakage of some part of the plane or of its essential accessories. A considerably higher percentage are due to bad weather—ice, fog or storm.

greatly improved and is now being manufactured by the Gaertner Scientific Corporation, of Chicago. As manufactured by the company the instrument has been still further improved. One of the earlier improved models was described in a previous number of the ARCHIVES.[2] In 1933-1936 this model of the instrument was used by Lieutenant-Commander C. J. Robertson, of the United States Naval Service, for the study of fitness for aviation on such points as entrance requirements, disqualification for the service on account of age, fatigue in relation to the number of hours in the air, individual susceptibility to fatigue and other points. Dr. Robertson has published his results in a series of articles in the *United States Naval Medical Bulletin* and the ARCHIVES.[3]

With this instrument can be measured, among other things, the speed of change of adjustment of the eyes for clear seeing at near to clear seeing at far and back again to near. This involves a measurement of the speed of vision, the speed of use of the muscles of the eyes in the perfect coordination needed for the clear seeing of a small detail and the speed of accommodation. It constitutes an extremely sensitive test of the ocular and oculomotor fitness of the aviator and of small disturbances in this fitness and also a delicate and effective test for bodily and mental fatigue and other disturbances in physical and mental facility and proficiency. Fatigue, for example, has to be tested through its effect on some function. Perhaps no more delicate means can be found for detecting fatigue than observation of its effect on speed in those uses of the eyes which require highly coordinated changes in muscular adjustment. The delicacy and accuracy of coordination that are required in these adjustments will be realized when one remembers that changes in the convergence of the eyes are made by six pairs of muscles which serve to support as well as to move the eyes, and that the breadth of the images on the two retinas which must be combined into one in seeing is for the standard test object of the order of thousandths of a millimeter. Also, in changing the vision from near to far and back again to near, the muscles of accommodation must act in perfect coordination with the muscles that move the eyes. Still further, the sensorium must function at a high level of efficiency.

2. Ferree, C. E., and Rand, G.: An Instrument for Measuring Dynamic Speed of Vision, Speed of Accommodation and Ocular Fatigue, Arch. Ophth. **15**:1072 (June) 1936.

3. Robertson, C. J.: Measurement of the Speed of Adjustment of the Eye to Near and Far Vision, U. S. Nav. M. Bull. **32**:275 (July) 1934; A Comparative Study of the Measurement of the Speed of Adjustment of the Eye for Near and Far Vision, ibid. **33**:187 (April) 1935; Measurement of Speed of Adjustment of Eye to Near and Far Vision, Arch. Ophth. **14**:82 (July) 1935; Measurement of Speed of Adjustment of Eye to Near and Far Vision: A Further Study, ibid. **15**:423 (March) 1936; Effect of Fatigue on the Adjustment of the Eye to Near and Far Vision, ibid. **17**:859 (May) 1937.

The test is without doubt one of the most sensitive that has ever been devised for the detection of any imperfection in the oculomotor, accommodative or sensory functions or any temporal disturbance in these functions. It is with these temporal disturbances that we are particularly concerned in this paper. Common causes of these disturbances are fatigue, loss of sleep, worry or any mental state which distracts attention, the variations in physical and mental efficiency and alertness common to every one in the course of time and illness. Any of these may be sufficient to cause the aviator to fail or falter at a critical time in the high degree of service that is required of him. The profound effect of fatigue and other disturbances of bodily and mental efficiency on such highly organized and delicate muscular coordinations as are required in the speedy use of the eyes is too well recognized to need further mention here.

It seems strange that so much care is taken to see that the plane is in perfect condition before a flight is undertaken and so little attention given to the condition of the aviator. While it is true that a human being cannot be treated as a machine, it is known, as has already been noted, that he is subject to many disturbances from day to day that render him unfit for services which require supernormal fitness and proficiency and involve responsibility for human life and safety. It seems only reasonable, therefore, that the fitness of the aviator, as well as the fitness of the plane which he operates, should be tested before each flight is undertaken. It is surely not enough to require only an entrance test of fitness and then allow him to go on without further check, even without regulation of his conditions of living, until age or some mishap retires him from service.[4]

Our personal feeling is that a test of each aviator should be made immediately before and after each flight. We offer the following reasons for this:

1. The test before the flight can be used to prevent the aviator from going into the air when he is clearly and dangerously unfit for service. It is neither fair nor good public policy that knowledge of

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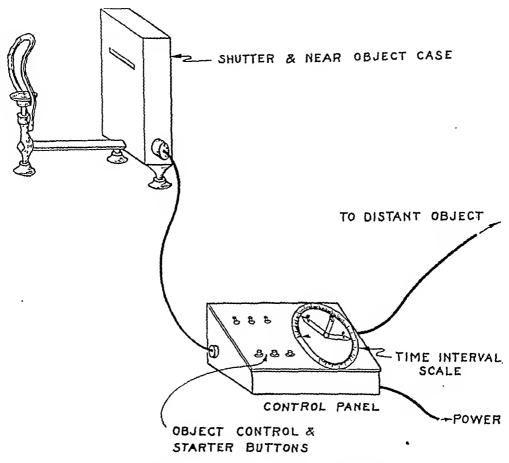
4. We understand that some improvement in this respect has already been made or is in contemplation. The following statements, for example, are quoted from the article by Major-General Fechet referred to earlier in the paper: "Striving to promote continued pilot fitness, we developed a new profession in the Army—that of Flight Surgeon. He has paid us handsome dividends. We found that annual or semi-annual examinations were not enough. We needed a smart medico to keep the pilots under daily observation. . . . Flight Surgeons paid off in reducing airplane crashes. I commend that thought to commercial air-line operators." He said further: "Health is mental as well as physical. I think the mental side plays a bigger rôle in air pilot health than the physical. A man who is worried and preoccupied about domestic discord or financial extremities may be more unsafe than one subject to fits or fainting spells."

his fitness should depend on his own report. In combat flying, in particular, he might well be prevented from making such a report through fear of being called a slacker or because of patriotism or personal pride. In commercial service, too, many reasons might operate to deter him from making a report of unfitness. The responsibility for making such report should be taken out of his hands and consigned to a competent examiner. A surgeon, however long his experience and however well demonstrated his ability, volutarily subjects himself to a test of steadiness of hand and keenness of eye before undertaking a critical operation. Surely in these offices requiring services equally responsible for life and safety there should be some test of fitness immediately before the service is undertaken. Such precautions might be considered extreme had it not been so clearly demonstrated that something is radically wrong in modern aviation.

2. The test at the end of the flight would indicate how well the aviator has stood the strain of his service. It would give valuable information as to his susceptibility to fatigue and make it possible to assign him to the length and kind of service he is capable of performing. It would also give a great deal of valuable general information as to the number of hours in the air and the amount of strain which aviators, taken collectively, can reasonably be expected to stand.

3. From the results of the tests, graphs or curves can be plotted which will give a splendid picture of the aviator's fitness, his endurance, his susceptibility to fatigue, the consistency of his service and other points. In short, these records would serve as the basis for a high type of personnel service in aviation. From these graphs it can also be readily seen when the aviator is becoming incapacitated for service through age or some other cause. This alone should be a sufficient reason for adopting some such program.

4. A feasible test with a suitable instrument is available. The test does not require more than ten minutes to perform, and the result can readily be given a numerical rating. The instrument is easy and convenient to operate, and the entire program is well within the technical capabilities of the average flight surgeon:

Two forms of the test may be suggested: (1) The time required for the discrimination of the object at near and for the change to far and back again to near may be measured in each test, or (2) in a series of preliminary tests the median or average time required for these performances may be determined for each aviator and taken as his standard of performance. In the routine procedure of testing, the instrument should be set to give these times of exposure. The test may consist of ten or some suitable number of trials to ascertain in what percentage of cases the aviator can attain his standard of performance. This percentage may be accepted as the index of his fitness at that time.

The instrument recommended may be called a multiple exposure tachistoscope. As described in the previous issue of the ARCHIVES,[2] it comprises, it will be remembered, a driving mechanism, four sectored disks so arranged and of such size as to expose in immediate succession, in turning, a near test object on the left, a far test object in the median plane and a near test object on the right. The test objects are the letter E, the openings of which can be turned in eight different directions to give an objective check on the judgment. The far test is provided

An electrical multiple exposure tachistoscope.

with a remote control such that these adjustments can be made by pressing an electric key at the position of the examiner. The distance of the far test surface and the lateral separation of the two near test surfaces can be varied at will. The sectored disks are turned by means of a constant speed motor provided with gears to give suitable reductions in speed and an intermittent gear which causes the disk covering the test objects prior to the beginning of the exposure to stop in exactly the same position at the end of a single rotation. In the preferred form the values of open sector are adjusted by small worm gears which serve both to change the position of the movable disks and to hold them firmly in position for any given setting. Exposure may be made in

continuous series up to four seconds. Both the angular values of open sector and the times of exposure can be read from suitably positioned, graduated scales.

A later form of the instrument is an electrical tachistoscope. This is the form now being manufactured by the Gaertner Scientific Corporation. In the figure are shown the control panel and the case containing the near test objects, the means for illuminating these test objects and the shutters for exposing both the near and the far test objects. The shutters are operated electromagnetically and are so arranged as to expose in sequence the test objects in the order already noted, namely, the near test object on the left, the far test object in the median plane and the near test object on the right. In the control panel are housed relay devices for operating the shutters and adjusting the position of the far test object. On the outside of the panel are provided suitable buttons and switches for these controls and a dial or time interval scale with hands that can be set to give the values of exposure desired. This form of the instrument has been designed especially to give compactness of construction and the maximum convenience of operation.

Either of the aforementioned forms of the instrument makes possible the following determinations: (*a*) the use of a set of very sensitive tests, which take into account as no other tests do both the motor and the sensory functions of the eyes in just the proportion that they occur in the act of seeing objects in different directions and at different distances; (*b*) the testing of the dynamic speed of vision with either the oculomotor or the accommodative feature emphasized (in the dynamic test for speed of vision the eyes are required to shift their regard from one object to another or to a series of objects and to discriminate them in turn during the time or times of exposure; these conditions test not only the speed of reaction of the sensorium but also the oculomotor facility and proficiency), and (*c*) the measurement of the time required to change from near to far and from far to near in combination or separately.

The test with this instrument has the following practical uses: (*a*) It is a means of detecting abnormalities and depressions in the oculomotor functions in the work of the clinic. (*b*) It constitutes a test for vocational fitness in all cases in which dynamic speed of vision is an important requirement. (*c*) It may be used as a limiting test for age. (*d*) It is a means of measuring ocular and oculomotor fatigue and recovery and of testing individual susceptibility to ocular fatigue and the capacity to recover. Because of the profound effect of fatigue and other disturbances of bodily and mental efficiency on such highly organized and delicate muscular coordinations as are required in the speedy use of the eyes, the test may be used also as a very sensitive means of detecting bodily and mental fatigue and other disturbances

in physical and mental proficiency which occur frequently in the normal course of living. It may be used also for detecting and studying the effect of change of altitude, temperature and other variations in the physical conditions to which the aviator is subjected. (*e*) It may serve as a means of training eyes to greater oculomotor and accommodative facility.

SUMMARY

With the growing conviction that the pilot is an important factor in the increasing number of airplane crashes, it seems that more attention should be paid to fitness in the selection of pilots and to making sure that they are fit for service at all times when they are called on to render service. It is strange that so much care is taken to see that the plane is in perfect condition before a flight is undertaken and so little attention is given to the condition of the pilot. While it is true that a human being cannot be treated as a machine, it is known that he is subject to many disturbances from day to day that render him unfit for services which require supernormal fitness and proficiency and involve responsibility for human life and safety. These disturbances can be shown by test.

In the paper a very sensitive test for these disturbances in fitness is proposed, and a convenient instrument for giving the test is briefly described. The test falls well within the technical capabilities of the average flight surgeon and should not require more than ten minutes to perform. It is recommended that the test be given both immediately before and immediately after each flight. The reasons for this are as follows: 1. The test before the flight can be used to prevent the aviator from going into the air when he is clearly and dangerously unfit for service. It is neither fair nor good public policy that a knowledge of his fitness should depend on his own report. The responsibility for making such a report should be taken out of his hands and consigned to a competent examiner. 2. The test at the end of the flight would indicate how well the aviator has stood the strain of his service. It would give valuable information as to his susceptibility to fatigue and make it possible to assign him to the length and kind of service he is capable of performing. It would also give a great deal of valuable general information as to the number of hours in the air and the amount of strain which aviators, taken collectively, can reasonably be expected to stand. 3. From the results of the tests, graphs or curves can be plotted which will give a splendid picture of the aviator's fitness, his endurance, his susceptibility to fatigue, the consistency of his service and other points. From these graphs it can also be readily seen when the aviator is becoming incapacitated for service through age or some other cause.

NEAR REACTION OF THE PUPIL IN THE DARK

A QUANTITATIVE STUDY

F. HERBERT HAESSLER, M.D.

MILWAUKEE

Although pupillary reactions have been thoroughly (one would be tempted to say exhaustively) studied, I have found no reference to a publication of data on the effect of adjustment for near vision acting alone as a stimulus to pupillary contraction. As is well known, the pupil contracts when the illumination of the eye is increased and also when the pair of eyes is adjusted for near vision. To study the character of the latter reaction alone it is necessary to observe the effect on the size of the pupil of near vision in darkness. This was accomplished as follows: In a totally dark room the subject of the experiment fixed his gaze on one of the luminous characters on the dial of a radiolite watch which was held at a measured distance from the eye and in such a position that the angle between the axis of the eye and the camera remained constant throughout. The shutter of a previously adjusted camera was then opened, and a flash bulb the illumination time of which was less than one-tenth second was ignited. It is known that there is a latent period of one-fifth second in the pupil's reaction to light; hence the picture records the size of the pupil which results from the near adjustment only. That the light of the radiolite watch is below the threshold of the pupil for light can be demonstrated by the entoptic pupillary phenomenon. Figure 1 makes clear the practical arrangement of this simple apparatus, and figure 2 shows how a constant angle can be maintained between the axis of the eye and that of the camera.

The apparatus consists of a head and chin rest (*a*), a Leica camera (*b*), a socket for the flash bulb (*c*), and a card with the fixation object *e*, movable on the bar *d*. The bar *d* has notches in it, which can be felt in the dark, so placed that the angle between the visual axes when the eye fixes an object at these points will be 6, 12, 18, 24 and 30 degrees, respectively. It was assumed that the center of rotation of the eyeball is 12 mm. behind the corneal apex, and a separate stick was prepared for each subject, the measurements being based on the interpupillary distance.

In each experiment the subject placed his head firmly in the head and chin rest. The camera and the rod carrying the fixation object were

adjusted satisfactorily. The room was darkened, and five minutes was allowed for the eyes to become dark adapted. A picture was then taken with the eyes focused on the object located at a distance such that there was an angle of 6 degrees between the visual axes. All further manipulations were easily performed in the dark. After two or three minutes, when the after-image of the flash-light had paled, the radiolite object was moved to the next notch, and a flash-light photograph of the same eye was taken again. This process was repeated until photographs were obtained of the pupil in the state which it assumed when the angle between the visual axes was 6, 12, 18 and 24 degrees, respectively. These distances were chosen in the belief that the near reaction of the
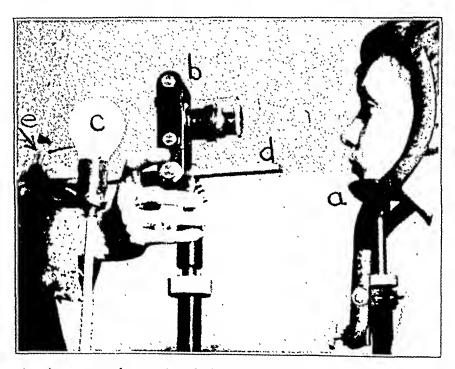
Fig. 1.—Apparatus for study of the near reaction of the pupil in the dark. *a* is the head and chin rest; *b*, the camera; *c*, the flash bulb, and *e*, the fixation object, movable on the bar *d*.

pupil is probably associated with convergence rather than with accommodation. If this is true the graphs for the two processes could be represented by straight lines, which, with the introduction of a suitable constant, would be parallel. The graph showing the accommodation for these distances expressed in diopters is a curved line, but it differs so little from the graph for convergence that it is not possible to correlate the curves for the pupillary changes with one or the other of them. The pupillary and corneal diameters were measured directly on a projection from the film enlarged so that the image of the cornea was from 8 to 10 mm. in diameter.

All the data gathered are presented. No photographs were ignored except the first few taken in experiments to try out the technic. Once the series was started, all the data were considered. The subjects were a boy of 11 years, two girls aged 12 and 13, respectively, and a woman aged 36. In refraction carried out with the pupils under the influence of homatropine the refractive error was found to be less than 0.5 D. The subjects had no hyperphoria, and their eyes could converge beyond the point necessary to make the angle of 24 degrees. Inspection of the data makes it clear that the decrease of the pupillary diameter is proportional to the increase in the angle of convergence in twenty-seven
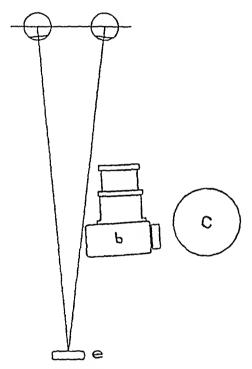
Fig. 2.—Diagram illustrating the principle of the apparatus. *b* is the camera; *c*, the flash bulb, and *e*, the fixation object.

of the forty experiments. The figures in the third, fourth, fifth, sixth and tenth columns state the measurement in millimeters of the pupillary and corneal diameters on the projected image. In the seventh, eighth and ninth columns the decrease in the pupillary diameter from step to step is recorded in millimeters as measured on the projection. In the eleventh and twelfth columns are stated the maximum and the minimum pupillary diameter in millimeters and tenths of a millimeter, as calculated from the assumption that the actual corneal diameter of all the eyes was 11 mm.

In the twenty-seven series of photographs of the pupil which I have considered successful, no one increment of shortening differs from the

average of the series by more than 20 per cent. As the pupillary circumference is proportionate to the pupillary diameter, the decrease in the pupillary diameter represents shortening of the fibers of the sphincter pupillae. Inspection of the table reveals no uniform tendency among the thirteen series of photographs that differ from the majority; there-

Data for Experiments

Experiment	Subject	Difference Between Relative Pupillary and Corneal Diameters, Mm.				Increment of Change, Mm.			Relative Corneal Diameter, Mm.	Pupillary Diameter, Mm.		Normal Near Reaction
		6°	12°	18°	24°					Maximum	Minimum	
1	H	49.0	45.5	42.0	38.0	3.5	3.5	4.0	80	6.7	5.2	+
2	H	47.5	44.0	40.0	36.0	3.5	4.0	4.0	78	6.7	5.1	+
3	L	43.0	38.5	35.5	35.5	4.5	3.0	0.0	78	6.1	5.0	..
4	L	39.0	36.5	31.0	28.0	2.5	5.5	3.0	79	5.4	4.9	..
5	L	42.0	40.0	34.0	31.0	2.0	6.0	3.0	79	5.8	4.3	..
6	H	47.5	43.5	39.5	35.5	4.0	4.0	4.0	80	6.5	4.9	+
7	H	48.5	45.0	41.0	37.5	3.5	4.0	3.5	76	7.0	5.4	+
8	H	48.0	44.5	41.5	38.0	3.5	3.0	3.5	76	6.9	5.5	+
9	H	51.0	47.0	42.0	32.0	4.0	5.0	10.0	79	7.1	4.5	..
10	L	44.0	35.0	31.0	29.0	9.0	4.0	2.0	80	6.0	4.0	..
11	L	40.0	36.0	32.0	28.0	4.0	4.0	4.0	78	5.6	3.9	+
12	A	50.0	46.0	42.0	38.0	4.0	4.0	4.0	80	6.9	5.2	+
13	A	46.0	43.0	41.0	38.0	3.0	2.0	3.0	79	6.4	5.3	..
14	H	46.0	43.0	40.0	36.5	3.0	3.0	3.5	80	6.3	5.0	+
15	D	38.0	35.0	32.0	29.5	2.5	3.0	2.5	81	5.2	4.0	+
16	D	38.0	33.5	29.0	25.0	4.5	4.5	4.0	80	5.2	3.4	+
17	D	36.0	32.0	28.0	24.0	4.0	4.0	4.0	80	4.9	3.3	+
18	H	55.0	50.0	45.0	40.0	5.0	5.0	5.0	92	6.6	4.8	+
19	H	51.0	46.0	41.0	36.0	5.0	5.0	5.0	78	7.2	5.1	+
20	H	51.0	47.5	43.5	40.0	3.5	4.0	3.5	78	7.2	5.6	+
21	A	50.0	47.0	44.0	40.5	3.0	3.0	3.5	78	7.0	5.7	+
22	A	49.0	47.0	45.0	36.5	2.0	2.0	8.5	78	6.9	5.1	..
23	A	49.0	46.0	43.0	39.5	3.0	3.0	3.5	78	6.9	5.5	+
24	D	35.0	30.5	27.0	24.0	4.5	3.5	3.0	74	5.2	3.6	..
25	D	36.0	36.0	32.0	26.0	0.0	4.0	8.0	74	5.3	3.9	..
26	L	44.0	41.0	38.0	35.5	3.0	3.0	2.5	78	6.2	5.0	+
27	L	42.0	38.5	35.0	32.0	3.5	3.5	3.0	78	5.9	4.5	+
28	L	42.5	39.5	36.0	32.0	3.0	3.5	4.0	78	6.0	4.5	+
29	D	37.0	31.5	26.0	24.0	5.5	5.5	2.0	74	5.5	3.6	..
30	L	41.0	38.5	36.0	29.0	2.5	2.5	7.0	78	5.8	4.1	..
31	D	38.0	34.5	31.0	27.5	3.5	3.5	3.5	74	5.6	4.1	+
32	D	36.0	33.0	30.0	27.0	3.0	3.0	3.0	74	5.3	4.0	+
33	D	38.0	35.0	32.0	28.5	3.0	3.0	3.5	74	5.6	4.2	+
34	D	39.5	36.0	33.0	29.5	3.5	3.0	3.5	75	5.8	4.3	+
35	D	41.0	37.5	34.0	30.5	3.5	3.5	3.5	77	5.8	4.3	+
36	L	40.5	36.0	33.0	28.0	3.5	3.0	5.0	78	5.7	3.9	..
37	L	39.0	36.5	34.5	30.0	2.5	2.0	4.5	78	5.5	4.2	..
38	A	50.0	45.5	41.5	37.5	4.5	4.0	4.0	80	6.9	5.1	+
39	A	48.0	43.5	39.0	35.0	4.5	4.5	4.0	80	6.7	4.8	+
40	A	49.0	45.0	40.5	36.0	4.0	4.5	4.5	80	6.6	4.9	+

fore one can conclude, I think, that the twenty-seven series which are similar within the limits stated represent a description of the normal near reaction of the pupil in the absence of light.

SUMMARY

By means of instantaneous photographs of the pupils of the eyes that are in a known state of convergence in the dark, measurements were obtained which describe the pupillary reaction that is associated with near vision in the absence of other stimuli.

ABSTRACT OF DISCUSSION

Dr. Francis H. Adler, Philadelphia: Dr. Haessler has studied the effect on the pupil of the near point reaction, that is, the combined action of accommodation and convergence for objects placed at known distances in front of the eye. He has undertaken these measurements with the retina partially dark adapted. In this respect these measurements are new, and the data constitute interesting information.

A number of authors have previously studied the effect of accommodation acting alone on the contraction of the pupil under conditions of illumination varying from 200 to 0.5 lux. One author studied the contraction of the pupil at different distances when the light was kept constant at high intensities of illumination and then subsequently studied the same reaction when the illumination was stepped down.

Dr. Haessler has carried this experiment one step further by reducing the illumination to practically zero and studied the near point reaction in a dark adapted retina. The results give information as to the effectiveness of accommodation and convergence on the tone of the pupil when all light is excluded from the eye, and, as I read his figures, they appear to prove that even when light is excluded from the eye the effect of accommodating and converging for a known distance, such that the angle between the visual axis is 6 degrees, is sufficient to exert a tone on the pupil to contract it somewhat, because his figures at the start of the test show a pupil of approximately 6 mm. in diameter, whereas the diameter of the pupil of a nonaccommodating and nonconvergent eye under similar conditions of dark adaptation should be higher than this.

One can be certain that convergence was carefully controlled in his measurements, because he gives the actual angle between the visual axes. One cannot be so certain that accommodation was equally carefully controlled.

I could not predict how much accommodation the subject was actually making when fixing on the radiolite dial of the watch at known distances. I wish to know whether the subject was required at each test to tell the figures on the watch and if the figures on the watch were sufficiently small to require an accommodative effort.

I would not agree, therefore, that what was found is due to convergence alone, but perhaps that is a minor and more academic question, since, as Hess and others have shown long ago, this reaction of the pupil is an associated reaction and not a reflex, and under experimental conditions both convergence and accommodation can be isolated, and each is successful in causing contraction of the pupil.

Dr. Frederick H. Verhoff, Boston: I should like to ask Dr. Haessler if he made any attempt to see whether adduction alone has an effect on the pupillary reaction. I do not see how any one could determine whether accommodation alone or convergence alone has an effect on the pupil, because I know of no experiment by which one can separate these two functions. Even if one covers one eye and looks at a near object, convergence occurs. If Dr. Haessler or any one else has a method of separating the effects of these two functions I should like to know what it is. In other words, it seems to me impossible to determine whether the pupillary reaction comes primarily from convergence or from accommodation.

In regard to adduction, it would be interesting to know whether or not the mechanism for this is entirely distinct from that of the mechanism for voluntary convergence, and it seems to me that Dr. Haessler might have been able to obtain data on this question by producing convergence by prisms and ascertaining whether or not the pupillary reaction differed under these conditions from that resulting from normal convergence.

Dr. F. Herbert Haessler, Milwaukee: Dr. Adler mentioned accommodation. An effort was made to be sure that the subject did accommodate clearly.

The figures on the watch are very small. The subject's attention was constantly called to a definite number, the implication being "You are sure you see the number?" The reply was: "Yes, I see the number." Also, in the experiment with the greatest angle of convergence, namely, that around 24 degrees, if one tries to increase that angle of convergence, in other words, bring the watch nearer to the eye, the subject can no longer accommodate. Under such conditions the subject complained of blurring of a very small image.

Dr. Verhoff's suggestion about separating accommodation and convergence within the limits of these experiments has not been followed. At the distances which give the angles of convergence chosen, the graph for accommodation is pretty close to that for convergence. It is not precisely a straight line, but the curve is so slight that the experimental data are too gross to enable one to decide this point. However, experiments have been reported in which the pupillary reaction has been shown to be definitely associated with the convergence reflex.

EQUAL ADVANCEMENT AND RECESSION OPERATION FOR HORIZONTAL STRABISMUS

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Based on the principle of equal advancement and recession of the opposing muscles for horizontal strabismus, the method of operation here described has been found dependable in giving accurately the planned alteration of position of the eyeball which preliminary examination and testing have shown to be desired. One muscle is to be receded the same number of millimeters that the antagonist is advanced. Because of the equal advancement and recession there is little or no tension on the fixing sutures during the operation or during the healing process; adhesion of the displaced tendons takes place at the site of the sutures. Following the described sequence of the steps of the operation contributes to securing this lack of tension of the sutures and consequently to the exactness of the result.

Measurement of the displacement of the muscles is made with calipers set for the desired distance in millimeters and used to measure both advancement and recession (fig. 1). Seven millimeters is the maximum distance practicable, and this is used only in cases of extreme deviation; if greater effect is needed it should be obtained by operation on the other eye. I have used only ordinary calipers with fine but blunt points, but those described by Dr. John Green, having one point sharp to anchor at the insertion while the other marks the sclera, would give additional accuracy.

The muscle to be advanced is exposed by a horizontal incision of the conjunctiva and Tenon's capsule, and its tendinous portion is freed from side attachments. A double-armed suture is placed exactly at the insertion of the tendon, its loop embracing the middle third of the width of the tendon together with a little of the superficial portion of the sclera (fig. 2 A). The two suture needles are passed under the upper and the lower border, respectively, of the tendon and through the tendon at the borders of its middle third at exactly the distance from the loop in the insertion measured by the calipers (fig. 2 B). The two sutures are then passed through the upper and the lower lip, respectively, of the incision in the capsule and conjunctiva at the level of the insertion and in line with the loop of the suture (fig. 2 C). The main advancement suture is in place before the muscle itself is disturbed, and its position can be precise (fig. 2 D). The advancement is now

interrupted for the purpose of doing the recession. This much of the advancement is done first because the deviated position of the eye gives the best possible exposure of the field of operation.
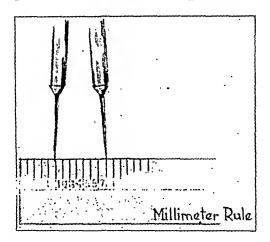
Fig. 1.—Calipers set for the desired change of position of the eye, to measure both advancement and recession; 7 mm. is the maximum distance.
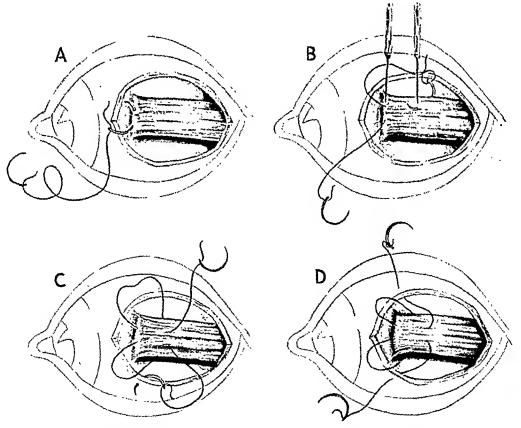
Fig. 2.—The muscle to be advanced is exposed by a horizontal incision of the conjunctiva and Tenon's capsule; a suture is placed at the insertion of the tendon, embracing the middle third of the tendon (A). The two suture needles are passed through the tendon at the borders of its middle third at the position measured by the set calipers (B). The two sutures are passed through the lips of the incision of the capsule and conjunctiva in line with the loop of the suture in the insertion (C). The main advancement suture is entirely in place before any alteration of position of the muscle (D).

The muscle to be receded is exposed by a horizontal incision of the conjunctiva and Tenon's capsule, freed from lateral attachments and raised on a thin and straight squint hook placed close to the insertion (fig. 3 *A*). The usual squint hook is too thick and too curved for this purpose. A double-armed suture is placed immediately behind the thin hook, its loop on the under-surface embracing the middle third of the width of the tendon, a little less than 2 mm. from the insertion. The two suture needles then are passed through the upper and the lower lip, respectively, of the incision in the capsule and conjunctiva, posterior to the suture loop, the distance being measured by the calipers (fig. 3 *B*).
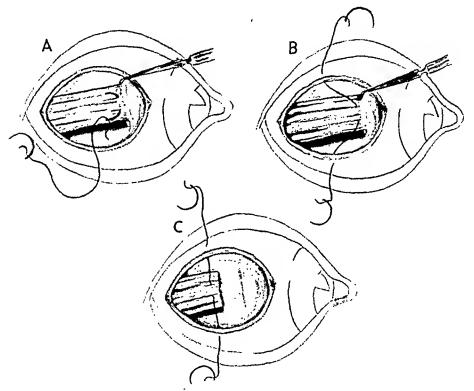
Fig. 3.—The muscle to be receded is exposed by a horizontal incision of the conjunctiva and Tenon's capsule; a suture is placed close to a thin hook, embracing the middle third of the tendon (*A*). The two suture needles are passed through the lips of the incision of the capsule and conjunctiva, the distance from the suture loop being measured by the calipers (*B*). The muscle, cut at its insertion in the sclera, retracts; the suture then is tied and secures the tendon to Tenon's capsule and closes the incision of the capsule and conjunctiva (*C*).

This measurement is made from the suture loop, not from the insertion. The tendon is cut from the sclera at the insertion, the hook remaining in place to protect the suture; this is done best with the Weber probe-pointed canaliculus knife. The severed muscle retracts, and the suture when tied unites the tendon with the capsule and conjunctiva (fig. 3 *C*). An additional suture may be needed to close the anterior

half of the conjunctival incision. It is essential that there be no vertical cuts or tears in the lips of the incision of Tenon's capsule, since the position of the retracted muscle during the process of healing depends on the integrity of the capsule between the suture and the capsule's anterior attachments. It is probable that adhesion of the tendon to the sclera almost always occurs finally; if not, the muscle is all the better for being lengthened rather than set back on the sclera.

The recession being completed, the advancement is resumed (fig. 4 *A*). A second advancement suture is placed in the superficial portion of the sclera, with a vertical loop, 2 or 3 mm. anterior to the insertion of the main suture (about half the measured advancement). The
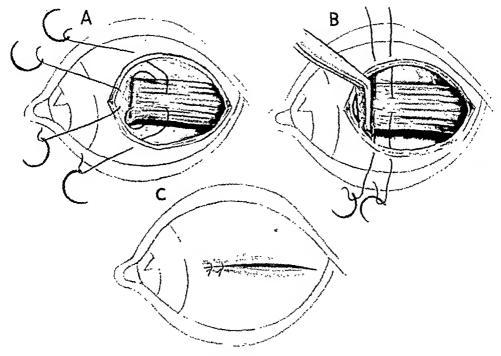
Fig. 4.—A second advancement suture is placed in the superficial portion of the sclera anterior to the main suture loop; the tendon, in the grasp of a muscle forceps, is severed between the forceps and the insertion (*A*). The tendon, in the grasp of the forceps, is elevated and drawn forward; the two needles of the anterior suture pass through the tendon at the borders of its middle third posterior to the forceps and then through the lips of the incision of the capsule and conjunctiva, being thus parallel to the main suture (*B*). The two advancement sutures, placed tandem and parallel, unite the sclera, tendon, capsule and conjunctiva and close the external incision (*C*).

tendon is grasped with a muscle forceps close to the insertion but with room left to cut the tendon between the forceps and the suture loop, care being taken to avoid cutting the suture; this too is done best with the canaliculus knife. The muscle in the forceps is elevated, and the two needles of the anterior suture are passed at the borders of its middle third, behind the forceps and 2 or 3 mm. anterior to the corre-

sponding part of the posterior suture, then through the upper and the lower lip, respectively, of the incision of the capsule and conjunctiva and again anterior to the corresponding part of the anterior suture (fig. 4 *B*). If the advancement is not more than 4 mm. a temporary suture loop can be used in place of the muscle forceps. The muscle is drawn forward to the advanced position; the sutures are tied, the posterior one being tied first, and the tendon end is tucked inside the incision after being trimmed with scissors if redundant. The advancement sutures, placed tandem and parallel, embrace in their loops the superficial portion of the sclera (the stump of the tendon is held in the posterior suture), the middle third of the tendon and both lips of the incision of the capsule and conjunctiva (fig. 4 *C*). The recession having been done, little traction is needed to draw the muscle into the position of advancement, and there is little tension on the sutures during the tying or during the healing. An additional suture may be needed to close the posterior half of the conjunctival incision. A binocular dressing is applied and maintained four or five days, with daily change for cleansing and use of any antiseptic which seems needed. Catgut sutures are left to absorb; silk sutures are allowed to remain undisturbed as long as eight days. General anesthesia is necessary for young children; for older children and for adults a 2 or 4 per cent solution of procaine hydrochloride injected along the two muscles is adequate.

CHEMISTRY OF THE RETINA

IV. THE BACILLARY LAYER

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CHICAGO

Little is known about the chemical nature of the visual purple, the chief light-sensitive substance in the retina. In the past the retina with its ten or more layers was generally extracted with a solution of digitonin or of sodium cholate to give a colloidal solution of visual purple which contained many other chemical substances naturally occurring in the tissue. The crude extract was then examined by optical methods to determine its photochemical properties. Recently the whole retina was extracted with solvents of lipids, and carotenoids were found. The controversial reports on the carotenoids gave only slight evidence of their chemical nature. It is questionable whether or not the light-sensitive substance and carotenoids come from the bacillary layer or the rest of the retina or from both.

This paper is a report on the attempt to isolate and to determine some of the chemical properties of purified visual purple. The experience which was gained in the investigation of the constitution [1] and of the lipids [2] of the retina was a great aid in the treatment of this light-sensitive substance.

EXPERIMENTAL PROCEDURE

Extraction of Visual Purple.—Fresh eyes from cattle from 1 to 2 years old were kept cold and were not exposed to light. The retinas without the retinal pigment epithelium were immediately removed under a 10 watt red photographic light and were then ground in a mortar and suspended in a cold 1 per cent solution of sodium chloride, 1 cc. of solution being used for each retina. After centrifugation at 2,100 revolutions a minute for fifteen minutes a layer of dark red visual purple formed a ring separating the water-soluble material from the insoluble residue. The red substance was removed by decantation, and the residue was again extracted. The slimy red substance was freed of water-soluble material by washing with a 2 per cent solution of sodium chloride at p_H 7.8. The yield of the red substance, now called crude visual purple, from moist bovine retinas was repeatedly found to be about 2.7 per cent, calculated on the basis of dry weight.

Serial extraction of the layer of visual purple obtained from 200 bovine retinas was made (475 mg. calculated as dry weight). With a 2 per cent solution of digitonin about 400 mg. of crude visual purple was extracted, which contained 300

1. Krause, A. C.: The Chemistry of the Retina: II. Chemical Constitution, Am. J. Ophth. **7**:555, 1936.

2. Krause, A. C.: The Lipids of the Retina, Acta ophth. **12**:372, 1935.

mg. of protein and 100 mg. of lipid. The material that was found to be insoluble after repeated extractions with solution of digitonin was extracted with a solution of sodium glycocholate until no soluble substance was obtained. The solutions of glycocholate contained 10 mg. of protein and 5 mg. of lipid. The residue was repeatedly extracted with a solution of sodium desoxycholate. The combined extracts contained 4 mg. of protein and 1.5 mg. of lipid. The insoluble residue consisted of a black pigment and a light gray gel. It contained 30 mg. of protein and 13 mg. of lipid. About 0.24 mg. of phosphorus was found in this lipid.

Visual purple dissolved in a 2 per cent solution of digitonin. About 1 mg. of sodium glycocholate and about 0.5 mg. of sodium desoxycholate, respectively, in aqueous solution dissolved 1 mg. of visual purple. Digitonin gave a pinkish red solution, sodium glycocholate a rose red solution and sodium desoxycholate a deep red solution, with the same concentration of visual purple. If the solutions are saturated with sodium or magnesium sulfate the visual purple is precipitated. A solution of sodium cholate dissolved little or no visual purple.

A series of samples each containing 1 cc. of a suspension of fresh visual purple layer which was equivalent to 19 mg. of the dried material were mixed with 1 cc. of a 2 per cent solution of digitonin, 1 cc. of a 2 per cent solution of sodium glyco-cholate and 1 cc. of a 2 per cent solution of sodium desoxycholate, respectively. After stirring for thirty-minutes, a red residue was found in the first two solutions. If the amount of red color of the solution of sodium desoxycholate was taken as 100, that of the solution of sodium glycocholate was 77 and that of the solution of digitonin was 43. However, repeated extraction removed the red color of the residues.

Analysis of Visual Purple.—A typical analysis of the crude visual purple, which was performed repeatedly, is described as follows:

The crude visual purple was separated from 123 Gm. of moist retina. It was repeatedly extracted with a 2 per cent solution of sodium glycocholate until the total volume of red extract was 50 cc. The gray gelatinous residue weighed 378 mg. and consisted of 308 mg. of protein and 70 mg. of lipid. The extract was half saturated with sodium sulfate at 25 C. A small white residue of lipoprotein, contain-ing 158 mg. of protein and 24 mg. of lipid, was removed by filtration. The solution was extracted with small portions of purified petroleum benzine (petroleum ether) until nothing further was removed. The extract contained 10.4 mg. of lipid, of which 4.7 mg. was cholesterol.[3] It did not react positively to antimony chloride or for the presence of glycocholate. The solution was saturated with sodium sulfate, and the visual purple was precipitated. The visual purple was exhaustively extracted with alcohol and then with ether. The white residue consisted of 294 mg. of protein. The orange-colored extract contained 142 mg. of lipid, 273 mg. of glycocholate and 2.2 mg. of phosphorus. The extract was evaporated to dryness and dissolved in 50 cc. of chloroform. The chloroform solution was evaporated almost to dryness, and then 100 cc. of acetone was added. The mixture was stirred with a stream of nitrogen. The precipitate from the acetone solution was washed with small portions of cold acetone. It weighed 150 mg. and contained 2.14 mg. of phosphorus, 60 mg. of glycocholate and, qualitatively, choline.[4] The phosphorus

3. Man, E. B., and Peters, J. P.: Gravimetric Determination of Serum Cholesterol Adapted to the Man and Gildea Fatty Acid Method, with a Note on the Estimation of Lipoid Phosphorus, J. Biol. Chem. **101**:685, 1933.

4. Lintzel, W., and Monasterio, G.: Eine Methode zur Mikrobestimmung des Lecithins in Blute und Plasma, Biochem. Ztschr. **241**:273, 1931.

and choline were derived from the lecithin. After saponification with a 0.4 per cent solution of sodium hydroxide at 25 C. for thirty minutes, 30 mg. of colorless fatty acids, 0.2 mg. of cholesterol and less than 1 mg. of unsaponifiable lipid were found. The saponifiable and unsaponifiable fraction of the residue gave no reaction to antimony trichloride.

The soluble acetone fraction was evaporated to dryness. It weighed 53.5 mg. After saponification with 0.4 per cent solution of sodium hydroxide at 25 C. for thirty-five minutes, the nonsaponifiable part weighed 31 mg. and contained 8.0 mg. of cholesterol as determined by the colorimetric method and 7.6 mg. as determined by the digitonin method. After the removal of the cholesterol by digitonin and then the removal of the digitonin, a biphasic test was made on the residue, of which about 8 mg. dissolved in the 92 per cent methyl alcohol and 5.5 mg. dissolved in purified petroleum benzine. The golden yellow epiphasic residue gave a positive reaction to antimony trichloride. The colorless hypophasic residue showed no color with antimony trichloride.

The epiphasic golden yellow pigment of the crude visual purple gave a lemon-colored solution with purified petroleum benzine and a light red color with chloroform. In other solvents of lipids the color deepened according to the refractive index of the solvent. The pigment behaved like a carotenoid hydrocarbon in that it absorbed iodine, gave a positive (blue) reaction to antimony trichloride, passed to the epiphase, contained no nitrogen and formed a layer on calcium hydroxide in the chromatographic tube.

From the spectroscopic measurements it was found that about 65 per cent of the golden pigment is in the layer of visual purple and about 35 per cent in the rest of the retina. Since the amount of the retina without the visual purple is relatively large and the crude method of separation of the visual purple removes only the densest red layer, some of the remaining retina is slightly pink. If the golden yellow pigment is considered to be in a concentration of 100 per cent in the layer of visual purple as it is removed, the relative concentration of the golden pigment in the remainder of the retina is 5 per cent. It is probable, then, that the golden yellow pigment comes wholly from the bacillary layer.

The saponifiable fraction contained a yellow lipid which was insoluble in water but at p_H 7.5 or above became soluble and colorless in an alkaline solution. Acidification of the alkaline solution liberated the yellow lipid again. It gave a positive reaction to antimony trichloride and had an odor more like that of clupanadonic acid than like that of geronic acid or its derivatives. This yellow lipid pigment behaved like the golden yellow pigment except that it was preferentially absorbed by calcium carbonate and was precipitated by barium and silver salts and decolorized by alkalis. No other lipid pigments or lipids giving a blue reaction to antimony trichloride were found with the exception of very small quantities of vitamin A.

Action of Light on Visual Purple.—The following analyses were made to determine the action of light on the visual purple:

A 50 per cent saturated solution of sodium sulfate containing visual purple previously extracted with purified petroleum benzine was filtered and exposed to sun-

light for thirty seconds until the red solution turned clear golden yellow and was then extracted with purified petroleum benzine; the extract gave a positive reaction to antimony trichloride. A solution of visual purple was extracted consecutively three times with purified petroleum benzine, giving 4.5, 1.6 and 1.2 mg. of lipid, which contained 1.93, 1.03 and 0.93 mg. of cholesterol, respectively. After extraction this solution on exposure to light and subsequent bleaching of the visual purple gave up to purified petroleum benzine 10.5 mg. of lipid containing 2.5 mg. of cholesterol. This lipid gave a positive reaction to antimony trichloride, in contrast to the negative reaction of lipid obtained from the petroleum benzine extract from visual purple kept in the dark. In other words, light liberated a lipid containing free cholesterol precipitable by digitonin and another lipid giving a positive reaction to antimony trichloride. The nonsaponifiable fraction of the soluble acetone extract of the visual purple, which was exposed to light, gave a slight reaction to antimony trichloride, and the quantity of nonsaponifiable lipid was equivalent to 26 mg. The lipid contained 7.4 mg. of cholesterol. The crude yellow acid lipid of the saponifiable acetone extract weighed 13 mg.

If the golden yellow solution which was first exposed to light thirty seconds and which was then extracted with purified petroleum benzine was illuminated two minutes more, it lost 70 per cent of its yellow color as measured colorimetrically, and a light yellow precipitate formed. The precipitate consisted of 22 per cent lipid and 78 per cent protein. After a short time the solution became colorless and clear. If the golden yellow solution was made acid with acetic acid, a yellow precipitate and a clear solution resulted. After the precipitate was washed with slightly acidulated water, the solid was extracted with cold alcohol, a white residue of protein and a yellow solution of lipid being left. The yellow precipitate obtained by precipitation in acid was found to be 27 per cent lipid and 73 per cent protein. Alkalis changed the golden yellow alcoholic solution of lipids to a pale yellow solution from which 20.4 per cent of the lipids were extractable with purified petroleum benzine. This nonsaponifiable fraction contained 50.5 per cent cholesterol as determined colorimetrically and gravimetrically, but no nitrogen or phosphorus. The reaction to antimony trichloride was negative.

The nonilluminated retina, from which the layer of visual purple was removed, was treated in the same manner as the layer of visual purple. It was first extracted with purified petroleum benzine. The lipid residue weighed 1.3 mg. It gave no reaction to antimony trichloride and contained 0.8 mg. of cholesterol. The acetone insoluble fraction was found by analysis to be composed of large amounts of nucleic acid, lecithin, other complex lipids and glycocholate. Of 2.69 Gm. of total solids from the retina, 56 mg. of lipid soluble in acetone was found. After the saponification of the acetone soluble fraction no yellow carotenoid behaving like an acid was found in the 30 mg. The reaction to antimony chloride of the saponifiable fraction was slightly positive. In the nonsaponifiable fraction of 26 mg., 19 mg. of cholesterol and 4 mg. of a lipid which gave a slightly positive reaction to antimony chloride were found.

These analyses have been repeated many times on bovine visual purple during the past two years, and no essential differences were found in the results or in the color of the products.

The manipulation of the retina and extraction were done under red light and in the cold. The solvents and other chemical compounds

which were used were purified by standard methods and kept in the dark. The alcohol was free from aldehydes and acids. The purified petroleum benzine, with a boiling point of from 30 to 60 C., was free from residue and unsaturated hydrocarbons. The salts of the cholic acids were recrystallized and the composition checked by analysis.

COMMENT

The visual purple seems to be a complex lipoprotein consisting of protein and a lipid behaving like a carotenoid. A second lipid, with the properties of a highly unsaturated acid, which may be a product of decomposition, was found in the crude visual purple. On exposure to light a golden yellow lipid with the properties of a hydrocarbon carotenoid is liberated from the lipoprotein. Further exposure to light changes the golden lipid to a colorless lipid which has no carotenoid properties. This lipoprotein, or the visual purple, seems to be found only in the bacillary layer of the retina. The liberation of free cholesterol by light may occur from the breakdown of other retinal proteins of the bacillary layer.

Although no beta carotene or other carotenoid was found in the retina, there is no reason why it should not occur here, particularly in the retinas of the Jersey or Guernsey breed of cattle after grazing in the pasture. The report of Brunner, Baroni and Kleinau,[5] and that of Euler and Adler [6] that beta carotene is the chief carotenoid forming a part of the visual purple was not confirmed. Wald [7] was also unable to find carotene or xanthophyll in the bovine retina. There is a good possibility that either of the yellow lipids may act like vitamin A in feeding experiments. The only explanation which can be offered at the present time for the inconsistent reports on the chemical nature of the visual purple is the extreme complexity and sensitiveness of the retinal tissue to heat and light.

There is every reason to believe that the colored lipid which forms a part of the visual purple may vary in chemical structure with the organism and that many kinds of visual purple may occur in nature. Perhaps the light-sensitive substance, which may be derived from a carotenoid and which may vary with the type of carotenoid taken as food, is modified by the tissue to correspond to the nature of the environment of the animal. Furthermore, it is probable that light-sensitive complex carotenoid derivatives occur in plant and nonretinal animal tissues.

5. Brunner, O.; Baroni, E., and Kleinau, W.: Zur Kenntnis des Sehpurpurs, Ztschr. f. physiol. Chem. **236**:257, 1935.

6. von Euler, H., and Adler, E.: Visual Purple, Ark. Kemi. Min. Geol. **11B**:6, 1933; cited in Chem. Abstr. **28**:2048, 1934.

7. Wald, G.: Vitamin A in Eye Tissues, J. Gen. Physiol. **18**:905, 1935.

CONCLUSIONS

The visual purple is a lipoprotein composed of a protein conjugated with a golden yellow lipid having the properties of a carotenoid derivative   There is a question whether or not the lipoprotein contains lecithin and cholesterol.   The action of light on the visual purple liberates the golden yellow lipid, which, on further exposure to light, breaks down into a colorless noncarotenoid substance.

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CHRONIC EDEMA OF THE CORNEA

REPORT OF A CASE

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The following case of unilateral edema of the cornea in a young adult of 30, unassociated with trauma, with ocular inflammation, infection or hypertension, or with any other apparent local or general disease, is reported because of the great rarity and the obscure nature and origin of the condition.

REPORT OF CASE

S. K., an intelligent and cooperative white man 30 years of age, a salesman, presented himself for examination at the clinic of the Brooklyn Eye and Ear Hospital, in the service of Dr. E. C. Place, on April 22, 1936. He complained of fogged vision of the right eye. The first visual disturbance was experienced in July 1932, while he was on a fishing trip. At that time vision became hazy but it cleared up entirely after a few hours of sleep. Hazy vision recurred at intervals up to December 1935. Since then the haziness had been present almost continuously. There was never any pain, irritation or sensation of a foreign body, redness of the eye, photophobia or lacrimation. There was no history of trauma. Rainbow halos around lights, of the type noted in glaucoma, were described. The visual impairment was more apparent on a cloudy day than a clear day, did not seem to change with the time of day, was not affected by worry or emotional upset and almost invariably appeared to be much less marked after the patient came out of the cinema.

The past history was not significant. The patient was married and had a normal child 5 years of age. He said he had never had a venereal disease. The general health had always been good; there were no gastro-intestinal complaints. There was no history of ocular or general disease in the family.

At the initial examination vision of the right eye was 20/40 and was unimproved with lenses; vision of the left eye was 20/15. The right eye presented normal adnexae; a white globe without any trace of conjunctival or pericorneal injection; dulness of the cornea, which on focal illumination with a Zeiss loupe was recognized as due to edema; a round pupil of the same size as its fellow and reacting actively to light, and an anterior chamber of normal depth. Corneal sensation, as tested with a fine wisp of cotton, was unimpaired. The tension, as measured with the Schiötz tonometer, was 19 mm. Slit lamp examination showed fine bedewing of the entire epithelium, which was most marked in the central area and similar to that observed in glaucoma; there were no deposits on the posterior corneal surface, nor was there any other evidence of inflammation. No staining with fluorescein was observed grossly or with the slit lamp. The left eye appeared to be normal in every respect, and slit lamp examination failed to show any corneal lesion similar to that in the right eye. With cycloplegia induced by homatropine hydrobromide retinoscopic examination revealed simple hypermetropia of low degree in

each eye. The red fundus reflex of the right eye with a plus 10 sphere was speckled, and the media other than the cornea and the fundus appeared normal in every respect. The peripheral and central visual fields and the blindspots showed no abnormalities.

Observed at intervals over a period of more than four months, the affected eye remained white and unirritated. Visual acuity fell to 20/70, corresponding to increasing corneal edema and loss of transparency. There never was any evidence of inflammation. There was no desquamation of epithelium. There was no corneal hypesthesia. The tension remained fairly constant at from 15 to 19 mm. (Schiötz), was uninfluenced to any appreciable extent by miotics or mydriatics, and showed little difference from that of the unaffected left eye. The lacrimal passages were permeable. There was no apparent lacrimal hyposecretion. Neither pupil dilated with the instillation of a 1:1,000 solution of epinephrine hydrochloride. On two occasions vision was found to be markedly improved after a stay of fifteen minutes in the dark room, once improving from 20/70 to 20/32, and the corneal cloudiness was observed grossly to have diminished. Atropine, pilocarpine, phenacaine, ethylmorphine hydrochloride and occlusion of the eye had no effect on the course of the edema.

On Sept. 2, 1936, the patient complained of pain, lacrimation and photophobia of the affected eye and presented on examination erosion of the epithelium and a large bulla of the lower portion of the epithelium. Vision was 20/100. The eye was anesthetized with butyn, and the entire epithelium was denuded to the limbus, the glistening Bowman's membrane being exposed. Vision now was 20/20. The fundus was clearly viewed and was normal in every respect. With the slit lamp three or four tiny grayish opacities were observed in the superficial portion of the stroma; there were no tears or folds in Descemet's membrane and no keratitis punctata, and the aqueous beam, which had on previous occasions been slightly clouded by the bedewed epithelium, was clear. Full strength tincture of iodine was applied to the entire surface of Bowman's membrane, and the eye was bandaged firmly. There followed a few months of recurrent erosions of the epithelium, which did not respond to the usual methods of therapy. The epithelium finally healed over but remained edematous, and the eye continued to be slightly painful and photophobic. The edema became gross and largely subepithelial, elevating the epithelium in the form of large bullae which were freely movable on pressure of the finger on the lower lid. The cornea at this stage was anesthetic. Vision was reduced to 10/200. On Feb. 4, 1937, the epithelium was again denuded, and a firm pressure bandage was applied. Microscopic examination showed sheets of well formed squamous epithelium; no keratinization was noted. The appearance of subepithelial edema persisted. Atropine sulfate, scopolamine hydrobromide, pilocarpine hydrochloride, phenacaine in solution of oxycyanide of mercury, pontocaine, thyroxin and cod liver oil were employed locally at various times, without benefit. A low fat (anticholesteremic), high calcium and vitamin-rich diet was given for several months, without apparent improvement.

On May 12 biomicroscopic examination by Dr. Milton Berliner revealed the following picture: With the finest beam the epithelium of the right eye was seen as a fine white line raised from Bowman's membrane by a dark area which may have contained fluid. This space (subepithelial) varied in depth, being deepest near the center of the cornea. There was a granular appearance of Bowman's membrane, which extended in places into the anterior third of the stroma. Definite folds of Descemet's membrane were seen, with many fine deposits of pigment on the posterior corneal surface. On retro-illumination the granular areas, seen arteriorly, took on the appearance of droplets (edema). On specular reflection

the folds were seen as dark lines over the endothelial cells. There were many crater-like depressions and fine dots, due to deposits in the posterior portion of the cornea. The appearance of the endothelium was similar to that seen in dystrophy. The corneal nerves were not enlarged; only a few were seen which were normal. The left eye was normal except for one or two annular superficial opacities resulting from old foreign bodies.

On May 17 the lids were sutured together. Ten weeks later the eye was perfectly white, painless and free from photophobia. The basic condition of edema persisted. There was a single, large, freely movable bulla that occupied the pupillary area, and the peripheral portion of the epithelium appeared finely bedewed.

A complete medical survey was made. General physical examination revealed a white man of 30 years, 5 feet, 5½ inches (166 cm.) in height and weighing 132 pounds (60 Kg.), without any gross physical defects. The blood pressure was 122 systolic and 80 diastolic. Examination of the nose and throat revealed a deviated nasal septum and residual tonsillar tags resulting from a previous operation; the paranasal sinuses transilluminated well. Roentgenograms of the sinuses showed no abnormality. Roentgenograms of the teeth disclosed an infected right lateral incisor tooth, which was extracted. Roentgenograms of the chest gave negative results. Urinalysis yielded negative findings. The Wassermann and Kahn tests of the blood were negative. Hematologic study showed 4,410,000 red cells, 82 per cent hemoglobin (Sahli), a color index of 0.93 and 12,000 white cells, with 61 per cent polymorphonuclear leukocytes, 31 per cent lymphocytes, 3 per cent eosinophils and 5 per cent monocytes. A later study of the blood showed 4 per cent eosinophils. The basal metabolic rate was + 2 per cent. The Mantoux test (carried out with from 0.1 to 1 mg. of old tuberculin) was negative. Study of the blood chemistry gave the following findings: a urea nitrogen content of 21.1 mg. per hundred cubic centimeters, a urea content of 45.1 mg., a sugar content of 95.5 mg., a calcium content of 9.6 mg., a phosphorus content of 4 mg. and a chloride content of 495 mg., an albumin content of 4.98 per cent and a globulin content of 2.31 per cent, a total cholesterol content of 333 mg. per hundred cubic centimeters in May 1936, of 222 mg. in June 1936 and of 310 mg. in February 1937, of which 85 mg. (or 27 per cent) was free. The dark adaptation of each eye when tested in September 1936 was normal, and that of the right eye when tested in March 1937 was again normal. On the latter date it was impossible to retest the affected eye. A thorough study with respect to allergy did not reveal any significant factor.

COMMENT

The essential feature in this case was edema of the cornea without any of the pathologic conditions which usually produce it. There was no associated gastro-intestinal or general disturbance. There was no urticaria. There was no history of trauma nor was there any sign of infection or inflammation. Under observation for four months the affected eye was white and unirritated, the epithelium did not desquamate, the corneal sensation was unimpaired and the ocular tension was not elevated. The symptoms of fogged vision and the rainbow colors around lights, as well as the appearance of bedewing of the corneal epithelium, suggested glaucoma, which was excluded by the normal tension, fundi and fields of vision. Recurrent erosions of the

epithelium were a complication. The edema progressed to a gross type, with the epithelial layer raised and forming large bullae. Biomicroscopic examination showed fluid under the epithelium and in the stroma, folds of Descemet's membrane and definite changes in the endothelium. The epithelium was entirely denuded on two occasions; after the first denudation tincture of iodine was applied, without benefit. Local therapy and dietary measures were likewise unavailing. Tarsorrhaphy was performed, which ameliorated the condition but did not remove the basic edema. A complete investigation, including consideration of endocrine imbalance, avitaminosis, allergy and a metabolic disorder as possible causative factors, did not yield positive results, with the exception of definite hypercholesteremia.

This condition falls into a class that forms a rather definite entity not previously described as such in the American or the British literature. Under "chronic edema of the cornea," "chronic intermittent corneal edema" and "chronic cyclic corneal edema" sixteen cases have been reported in the French and German literature. It occurs at any age from 10 to 70 years. Either sex may be affected. Heredity plays no rôle. One or both eyes may be affected. The symptoms are cloudy vision and rainbow colors around lights. There is no pain, photophobia, lacrimation or sensation of a foreign body; at most, there may be a sensation of fulness, roughness or warmth. The peculiar feature of the clouded vision is its variability: It may occur at intervals of days or weeks, with partial or complete subsidence, and there may be a regular daily cycle of greatest cloudiness on arising in the morning, which becomes less marked toward afternoon and subsides entirely toward evening. Not every patient presents this daily variation. There is no history of trauma. On examination there is no evidence of infection, inflammation, hypertension or any of the numerous possible causes of corneal edema. There is bedewing or vacuolation of the epithelium, which is most marked in the central area; the peripheral portion of the epithelium may be entirely normal. The corneal nerves may appear thickened and more clearly visible than in a normal cornea. Endothelial changes are usually demonstrable. Corneal sensation is not impaired. Desquamation of the epithelium is not a usual finding. Erosions of the epithelium, however, may occur. Elevation of the cholesterol content of the blood is a usual finding. The etiology is obscure, and there is no response to the usual methods of therapy.

Aubineau [1] first called attention to this entity in 1922 with a report of two cases, and he reported five additional cases in 1929. He credited

1. Aubineau, E.: Oedème cornéen et hypercholesterinémie, Bull. et mém. Soc. franç. d'opht. **35**:224, 1922; Ann. d'ocul. **159**:580, 1922; L' "oedème cornéen" des hypercholestérinémiques, Bull. et mém. Soc. franç. d'opht. **42**:283 (Aug.) 1929.

Despagnet[2] with describing the first case of this condition to be recorded. Löhlein[3] in 1913 described a case of fleeting corneal edema associated with urticaria; both phenomena were ascribed to the same unknown toxic cause. All Aubineau's patients had a history of gastro-intestinal or hepatic disturbance, and all showed hypercholesteremia. He ascribed the edema directly to the elevated cholesterol content, which he thought in some manner increased the power of imbibition of the cornea. With an anticholesteremic diet two patients were cured and the condition of one was ameliorated.

Marbaix[4] in 1932 reported a case of corneal edema of one eye associated with urticaria in a woman of 28 years. He found epithelial bedewing, clouding of the corneal parenchyma, thickening of the corneal nerves and endothelial changes. In addition, there was detachment of the endothelium in the same eye outside of the zone of edema and ascribed to injury sustained at birth. There was no response to diet or local therapy. He accepted the explanation of Aubineau and further suggested as a causative factor some toxic disturbance in the body resulting in both urticaria and corneal edema.

François[5] in 1933 reported in detail his observations in a case of corneal edema of the left eye in a man 52 years of age. There was no hypercholesteremia. There were a slightly elevated blood pressure and some renal involvement. Biomicroscopic examination revealed epithelial bedewing, clouding of the superficial and middle layers of the corneal stroma and thickening of the corneal nerves. He did not observe any changes in the deeper layers of the stroma, in Descemet's membrane or in the endothelium. He considered the condition to be a trophic disturbance and did a tarsorrhaphy, which resulted in complete restoration of the cornea to a normal status. The thickening of the corneal nerves suggested to him chronic neuritis of the cornea as the possible cause.

Hambresin[6] in 1934 reported a case of bilateral corneal edema in a man of 48 years who had been observed for a period of six years. There was slight hypercholesteremia. There were the characteristic epithelial bedewing and some alteration in the endothelium. An anticholesteremic diet had no effect. Sclerecto-iridectomy was performed on one eye, though the tension was not elevated, and the edema per-

2. Despagnet: Bull. Soc. d'opht. de Paris, 1888.

3. Löhlein: Selten Hornhautphänomen, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **39**:377, 1913; Rev. gén. d'opht. **33**:471, 1913.

4. Marbaix, M.: Un cas d'oedème de la cornée avec décollement probable de l'endothélium, Bull. Soc. belge d'opht., no. 64, April 1932, p. 117.

5. François, J.: L'oedème chronique de la cornée, Arch. d'opht. **50**:549 (Aug.) 1933.

6. Hambresin, L.: A propos d'un cas d'oedème cornée bilatéral, Bull. Soc. d'opht. de Paris, October 1934, p. 484.

sisted after the procedure. The edema seemed to disappear with the continued use of atropine sulfate and reappear with cessation of its use.

Haemmerli [7] in 1936 reported a case of typical chronic edema of the cornea, the patient being the youngest to be reported on, the onset dating back to the age of 9 or 10 years. Both corneas were affected. There was a definite cycle of cloudy vision. There were vacuoles in the epithelium, edema of the parenchyma and changes in the endothelium. All forms of therapy, both local and general, were unavailing. This author considered all the previous views as to the etiology of the condition untenable and the real cause unknown.

Blaauw, [8] in a clinical report of a case that he believed to belong to the group reported by Haemmerli, presented findings which were similar in many respects to those that I have recorded. There were recurrent erosions of the cornea, the formation of large freely movable bullae, marked clearing up of the edema after slit lamp examination in a dark room and lack of improvement after removal of the epithelium and the application of tincture of iodine. Cod liver oil locally applied seemed to have some salutary effect. Blaauw felt that the herpes virus played a rôle in the condition.

Graf [9] recently reported two cases with some interesting features. Each patient presented himself for examination at the very onset of the disturbance with a description of the typical daily cycle of varied vision, and in each the condition involved the left eye. In the first case, that of a photographer 32 years of age, there was a history of intermittent cramps in the thighs and legs, hepatic disturbance, slight hypercholesteremia and an eosinophil count of 9 per cent. Treatment with drugs, the roentgen rays and injections of insulin was of no benefit. The second patient, an optician aged 30, had a history of gonorrheal infection, excessive perspiration, hepatic disturbance, a normal cholesterol content of the blood and a positive Mantoux test. Autohemotherapy appeared to have some beneficial results. The condition was markedly improved after a period of observation of two years in the first case, and of one and a half years in the second case. The author remained skeptical as regards any form of therapy and stated that improvement occurred in spite of treatment. The principal observations on biomicroscopic examination were epithelial bedewing and vacuolation, changes in the size and visibility of the corneal nerves and endothelial changes. During the phase of greatest cloudiness of vision the epithelium pre-

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7. Haemmerli, V.: Das zyklisch auftretende Hornhautödem, Klin. Monatsbl. f. Augenh. **97**:745 (Dec.) 1936.

8. Blaauw, E.: Ueber das zyklisch auftretende chronische Hornhautödem, Klin. Monatsbl. f. Augenh. **98**:665 (May) 1937.

9. Graf, K.: Chronisches intermittierendes Hornhautödem als Ausdruck einer Angioneurose, Ztschr. f. Augenh. **91**:278 (March) 1937.

sented vacuoles of varied sizes, the corneal nerves at the level of the deeper layers of the corneal parenchyma were easily discerned and much enlarged, and the endothelium showed defects, black spots, loss of contour and roughness of surface. In the phase of diminishing cloudiness of vision the epithelial vacuolation was reduced to bedewing and and then to normality, the corneal nerves became pale and thin and disappeared entirely, and the endothelium tended to return to a normal appearance in the early stages. A diet rich in cholesterol increased the corneal edema, and vice versa. If the disease persists for any length of time the original condition of the endothelium is never restored; eventually the borders of the cells become obliterated, and their normal form disappears. The entire posterior wall of the cornea becomes more homogeneous. Between the cells black spaces, which persist for months, appear. When the disease is not brought to a standstill the entire posterior third of the cornea becomes opaque, and then the picture of the endothelium becomes completely obliterated. In the second case very tiny, ill defined deposits (considered to be proliferations of the endothelium) resembling the precipitates of uveal inflammation were observed on the posterior surface of the cornea. Graf regarded the condition as vasomotor trophic neurosis, an ocular manifestation of angioneurotic edema. The transient edema associated with the unexplained cramps in the thighs and legs, excessive perspiration, hepatic and gastro-intestinal disturbance, urticaria and eosinophilia support this contention. The edema results from seepage of aqueous into the corneal substance after changes in the endothelial layer, which, when normal, forms a barrier to the penetration of fluid. The thickening of the corneal nerves is the result of imbibition of fluid through the nerve sheaths. The endothelial changes result from direct injury of angiotrophic influence or are secondary to altered chemical composition of the aqueous. That the condition is trophic follows from the fact that the central portion of the cornea, which is farthest removed from the source of nutrition, is predominantly affected.

The subject of chronic corneal edema has not escaped record in the American literature. Graves,[10] under the title "Epithelial Degenerations," discussed cases in which the patients presented as symptoms colored rings around lights and misty vision, did not have glaucoma and showed on biomicroscopic examination detectable epithelial changes. He particularly pointed out that such changes may be "transient, or remittent, or unobtrusive yet persistent." Gifford,[11] in a paper on "The Mild Form of Epithelial Dystrophy of the Cornea," mentioned the cases of Aubineau as

10. Graves, B., in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 476.

11. Gifford, S. R.: The Mild Form of Epithelial Dystrophy of the Cornea, Arch. Ophth. 7:18 (Jan.) 1932.

apparently instances of the typical form of epithelial dystrophy, meaning Fuchs' dystrophy. In these cases of chronic corneal edema the clinical picture is definitely not that of Fuchs' dystrophy. In my opinion, these cases belong to the same group that Gifford described as cases of the mild form of epithelial dystrophy. Gifford specifically mentioned the typical daily cycle of variable vision, to which so much attention has been directed in the reports of cases of chronic corneal edema. The retention of normal corneal sensation and the absence of epithelial desquamation in cases in which the condition is uncomplicated, both of which are usual findings in chronic corneal edema, would seem to indicate that the epithelium is not intrinsically involved. There is little doubt that one is dealing with a primary disturbance of the endothelium, with secondary edema of the remainder of the cornea, in which the nerves partake. The undisturbed corneal sensation and the absence of pain militate against the conception of inflammation of the corneal nerves. The usual finding of hypercholesteremia is more than a coincidence and would seem to indicate a disturbance of metabolism. As in the case of all the corneal dystrophies, chronic corneal edema must be considered a nutritional or neurotrophic disturbance, but the basic etiology must remain for the present mere conjecture.

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AN INSTRUMENT FOR QUALITATIVE STUDY OF DARK ADAPTATION

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PHILADELPHIA

On several occasions I have presented data obtained by the examination of eyes for the study of dark adaptation.[1]

These tests were quantitative and required the instillation of a miotic, which was occasionally objected to by the patient. Then, too, the procedure was a time-consuming one, since it took about one-half hour for the actual study to be made and a like amount of time for the plotting of a graph.

In an extensive study of unselected cases one expects to find a large number of normal persons with normal dark adaptation. It was therefore felt that much time could be saved if a simpler device was used which would quickly eliminate this group of normal persons. With such a test only a single reading of the minimum light visible is taken, with the light as the constant and time as the variable. This is in contrast to the test previously used, carried out with a more complicated instrument, in which the time was the constant and the light was the variable.

A rapid study of the light threshold is also of advantage, since the number of cases requested to be studied by the other clinics in the hospital has been steadily growing. This is probably due to the increasing interest in the subject as a result of the vast amount of contributions to the medical literature lately in which dysaptation[2] has been associated with vitamin A deficiency resulting from acute infections, particularly those of the nasal mucous membranes, from diseases of the skin and blood or from renal calculi, and also in which tests for dysaptation have been recommended for the detection of deficiency of vitamin A of children in the schools. One of the practical applications of this rapid test for hemeralopia cannot be overemphasized. It is possible to decrease the number of accidents occurring at night by acquainting persons suffering from hemeralopia with their affliction. The eyes of these persons,
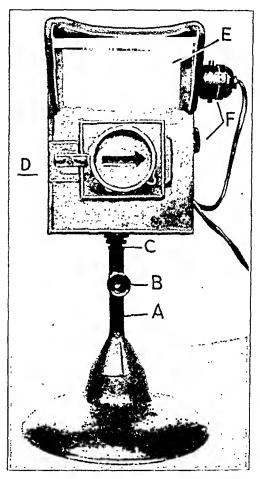
though they may be free from retinal pathologic changes, will indicate by a high light threshold as determined by the test that vitamin A deficiency is present.

The instrument for determining the light threshold (L. T.) [3] consists of a firm stand (*A*) in the tube of which is a thumb screw (*B*). The tube (*C*) of the lamp house (*D*) can be raised or lowered and

Instrument for the qualitative test of dark adaptation. *A* indicates the stand; *B*, the thumb screw; *C*, the tube of the lamp house; *D*, the dark adaptation chamber; *E*, the light adaptation chamber, and *F*, the switches. To use the instrument the light *E* is turned on for three minutes. This preexposure, which causes bleaching out of the visual purple, is then terminated, and the light in *D*, which illuminates the arrow, is turned on. In a normal eye the arrow should be seen and the direction which it takes should be recognized within a five minute period. Delay in seeing the arrow until five minutes or over classifies the condition as pathologic.

thus adjusted to suit the height of the particular patient and fixed in that position by the screw (*B*). In the lamp house (*D*) is a seasoned

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3. Other terms used are light minimum (L. M.), minimum visible (M. V.) and minimum light visible (M. L. V.).

light of daylight quality, constant in its voltage and illuminating the arrow or any other stimulus which it is desired to use. This arrow can be rotated at the will of the examiner without the knowledge of the patient. This helps to act as a check on the patient's veracity. Light adaptation is obtained with use of the lamp house (E). Switches (F) are used for illuminating the lamp houses for study of the light adaptation and the dark adaptation.

Since the saving of time is a factor, and as this study is only qualitative, no miotic to equalize the size of the pupils of every patient examined is necessary. A saving of from thirty to forty-five minutes is thus accomplished.

TECHNIC

1. The subject's vision is tested, and an ophthalmoscopic study is made. When the study of the dark adaptation is in the hands of a technician and the fundus cannot be examined, it may be assumed that in cases in which there is vision of 6/9 in each eye the maculae are intact.

2. All examinations for determination of the light threshold are made in a totally dark room.

3. The subject's forehead is placed against the frame of E for light adaptation for three minutes. A number of studies have been made as to the relative values of longer and shorter periods of light adaptation, as well as greater and lesser intensity of light in the light adaptation. The purpose of these studies was to obtain the effect which previous light adaptation had on subsequent readings for the light threshold.

The findings, however, brought out that a period of three minutes and a 100 watt light, used in my previous studies,[4] gave the most valid and reproducible results.

4. After three minutes the light is extinguished in the light adaptation lamp house (E) and switched on in the dark adaptation chamber (D), thus illuminating the arrow. The arrow is placed in the direction desired by the examiner.

The patient is moved back about 9 inches (23 cm.) from the arrow. Exact measurement of this distance is not necessary, as I have found that results have not been materially affected by slightly increasing the distance. The patient's hand is directed where the stimulus will be seen. This is done in order that the patient may not have to look in various wrong directions for the stimulus, with consequent delay in the time at which he actually sees the light.

Since time is the only important variable considered in this test, by indicating occasionally with the subject's hand where the light stimulus (the arrow) is to be seen the examiner fulfils a twofold purpose: 1. The patient is kept interested in the experiment. 2. Time is saved, and more valid results are obtained because the patient is looking in the right direction and can detect the light stimulus instantly. For a normal person the time that elapses from the termination of light adaptation to the recognition of the arrow and its position (the light threshold) should be under five minutes. A lapse of five minutes or over for reaching the light threshold is considered evidence of a pathologic condition, and the finding is grouped under dysaptation.

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4. Feldman,[1a] p. 1006.

RESULTS OF EXAMINATION

One hundred and sixteen subjects were examined. Except for excluding patients with glaucoma,[5] these subjects were unselected.

Forty-seven subjects showed a high light threshold, i. e, pathologic dark adaptation, or dysaptation. Of these, thirty-one were females. An equal number of males and females showed the normal light threshold.

Since in this test both eyes are examined at one time, it should be noted that even fewer subjects than those considered to have a normal light threshold would be classed as having this if each eye was examined separately.

Obviously, if one eye has a pathologic condition and the other is normal, the arrow would be recognized by the good eye and the subject

TABLE 1.—*Persons Who Had a Normal Light Threshold and a Pathologic Condition of One or Both Eyes*

No. of Subjects	Condition
2	Chorioretinitis, with one eye affected
1	Exudative choroiditis, with one eye affected
1	Old choroiditis, with one eye affected
1*	Choroiditis, with both eyes affected
1	Choroiditis, with one eye affected
1	Old choroiditis, with both eyes affected
1	Bilateral macular choroiditis
1	Choked disk
1	Sclerosing keratitis

* I have noted several cases of choroiditis in which there was normal dark adaptation (footnote 1b, table 2, p. 651). It seems that the opinion of Traquar (Percival, A. S.: Notes on the Light Sense, Tr. Ophth. Soc. U. Kingdom 42 : 285, 1922) should be qualified to read that diffuse choroiditis tends to cause dysaptation more than choroiditis localized in a small area does. The small area of localized choroiditis may, unfortunately, not be included in the area examined, when the study of the light sense is made.

would be classed as having a normal light threshold. A test of the visual acuity and examination of the fundi of each patient would, however, acquaint the examiner with the ocular disorder present. In cases of sympathetic involvement [6] of an eye with disease of the other eye there may be a high or pathologic light threshold. This is the only type of case, to my knowledge, in which examination of an eye with an apparently normal fundus will show dysaptation, and the disorder might be misconstrued as vitamin A deficiency.[6]

CASES OF NORMAL LIGHT THRESHOLD

Included in the group of persons with a normal light threshold, it will be noted, are several in whom one or both eyes show a pathologic

5. The reason for excluding glaucoma is that in most cases of chronic glaucoma the light threshold is pathologic, which would greatly increase the number of cases of dysaptation.

6. Feldman,[1a] p. 1014.

condition, as listed in table 1. I have classed the subjects listed in the table and the rest of those with a normal light threshold as persons with normal regeneration of the visual purple and therefore as persons who had a normal amount of vitamin A, sufficient to permit such regeneration to take place. The least time in which the arrow was recognized by a person in this group was thirty seconds, and the greatest, up to but not including five minutes.

CASES OF PATHOLOGIC LIGHT THRESHOLD

Persons who took five minutes or more for the light stimulus to be recognized were considered to have a pathologic light threshold. All

TABLE 2.—*Persons Who Had a Pathologic Light Threshold and a Pathologic Condition*

No. of Subjects	Condition	Age, Years	Vision Right Eye	Left Eye	Light Threshold Time
1	Hypertension—kidney disease	40	6/9	6/12	6 min.
1	Arteriosclerosis	65	6/9	6/9	7 min.
1	Old choroiditis of right eye................	49	6/9	6/9	6 min.
1	Arteriosclerosis	69	6/15	6/15	5 min.
1	Choroidal retinitis; incipient cataract; hemorrhagic choroidal retinitis; corneal opacity	48	6/60	6/60	6 min.
1	High myopie; choroiditis..................	33	6/60	6/30	7 min., 30 sec.
1	Arteriosclerosis	39	6/6	6/6	12 min.
1	Renal calculi	34	6/6	6/6	10 min., 15 sec.
1	Recent undiagnosed acute infection........	56	6/6	6/6	10 min., 30 sec.
1	Severe attack of grip 1 mo. previously.....	39	6/6	6/6	10 min., 45 sec.
1	Had 4 pelvic operations in last 2 yr......	32	6/6	6/6	5 min.
1	Arthritis of knee..........................	21	6/6	6/6	5 min.
1	Thyroid disease; basal metabolic rate +35 to +40..........................	29	6/9	6/6	13 min.
1	Uses microscope	43	6/6	6/6	12 min.
1	Complains of photophobia..................	53	6/9	6/9	12 min., 30 sec.
1	Complains of difficulty in seeing at night	17	6/15	6/60	8 min., 15 sec.
1	Vague pain of right side—possibly due to renal calculi	40	6/5	6/15	8 min.
1	Hypertension and gastric ulcer...........	38	6/6	6/6	8 min., 15 sec.
1	Works where there is sun glare...........	27	6/6	6/6	5 min., 30 sec.
1	Diabetes and high blood pressure.........	50	6/6	6.6	8 min., 15 sec.
1	Asthma, sinusitis and chronic mastoiditis	51	6/6	6/6	10 min., 15 sec.
1	Sinus infection for 3 yr...................	10	6/6	6/6	10 min., 15 sec.
1	High myopia *	28	6/60	6/12	10 min., 30 sec.

* Attention has previously been called to this condition as a cause of dysaptation (footnote 1).

the subjects examined in this group had normal fundi unless otherwise stated. Table 2 gives this grouping.

The rest of the subjects who showed a light threshold higher than normal varied in age from 14 to 70 years and had irrelevant histories. Their light threshold time varied from five minutes to ten minutes and forty-five seconds; in many cases vision was as good as 6/5. These subjects could be classed as persons with failure of regeneration of the visual purple due to vitamin A deficiency, without subjective symptoms. Included in this group would also be those already mentioned in table 2 whose light threshold was analyzed who did not have a pathologic condition of the fundus.

CONCLUSIONS

An instrument for a rapid qualitative check on the light threshold is described and its method of use given. In this test the only variable is the time at which the light stimulus is first noted by the patient.

A period up to five minutes is considered a normal interval for reaching a normal light threshold.

The light threshold time ranged from thirty seconds (in a subject with a normal light threshold) to twelve and one-half minutes (in a subject with an abnormal light threshold).

One hundred and sixteen patients were examined, about 40 per cent of whom showed dysaptation, nearly 70 per cent of these being females.[7]

An equal number of males and females showed a normal light threshold.

Dr. William Ezickson aided in this study by checking some of the patients.

Physicians Building.

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7. That females are more susceptible to dysaptation than males has been noted.[1a]

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

SURGICAL INTERVENTION FOR CATARACT
FROM THE PREOPERATIVE AND POSTOPERATIVE STANDPOINTS

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BALTIMORE

In this review I propose to take up the question of the survey of the case prior to operation for cataract, the choice of the operation to be performed and the most frequent postoperative complications and the way in which they may be combated.

SURVEY OF THE CASE

Of all the conditions which may mitigate against a successful outcome of an operation for cataract, that of a distant focus of infection is, in my opinion, undoubtedly the most frequent. Of such foci, that of diseased teeth is the one most frequently found. In the dental survey not only an inspection is desirable, but also a roentgen examination, since apical infections, abscesses and pyorrheal pockets are usually revealed by such a procedure. In case an apical abscess is revealed, extraction is the only satisfactory method of treatment, for although drainage and treatment may relieve the condition, the danger of a small area of infection remaining is too great to warrant subjecting the patient to this risk. When it is found necessary to extract teeth, because of either abscesses or pyorrheal conditions, sufficient time should elapse after this procedure before surgical intervention on the eye is attempted, in order to allow resolution to take place and the infection to be eliminated.

Steinbugler [1] questioned whether pyorrhea is ever a source of focal infection in ocular conditions, but if such a focus can cause arthritic conditions, certainly it could be a source of endogenous infection in the eye. The tonsils as a source of distant infection are not frequently at fault, although Jahnke [2] has seemingly shown conclusively a direct connection between septic tonsils and disease of the eye. At the age when cataracts are extracted, at least those of the senile type, the tonsils are usually atrophic (although this is not always the case), and therefore

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1. Steinbugler, W. F. C.: New York State J. Med. **32**:1223, 1932.
2. Jahnke, W.: Ztschr. f. Augenh. **72**:354, 1930.

they should be investigated as a routine measure. A diseased gallbladder is also occasionally a focus, but in such cases, as in cases in which a diseased appendix is the focus (Gresser [3]), the patient is usually aware of his condition, and if the infection is serious he will seek relief before surgical intervention on the eye is considered.

Syphilis in the latent form as a cause of complications following extraction of cataract was discussed by Ballantyne,[4] who expressed the belief that patients should have a preliminary Wassermann test, and treatment, if necessary. Hardy [5] has reported a case in which syphilitic parenchymatous keratitis developed after extraction of a cataract in a patient with a latent syphilitic infection. While postoperative complications are more frequent in the presence of latent syphilis, healing may be just as rapid in a person with syphilis as in the normal person and free from all complications.

Diabetes is probably the outstanding constitutional disease that causes postoperative complications. While the danger from a general anesthetic in cases of diabetes is of little importance to the ophthalmic surgeon, the danger of postoperative infections and postoperative hemorrhages is greatly enhanced in such cases. Thus, Wheeler,[6] in a survey of 2,123 extractions of cataract performed at the New York Eye and Ear Infirmary, found that hemorrhages occurred in 28.94 per cent of the patients with glycosuria, while the average incidence was only 5 per cent in cases of uncomplicated cataract. According to Benedict,[7] however, the hemorrhagic tendency in patients with glycosuria is not benefited by the use of insulin but is increased by it. He advised discontinuance of insulin for at least four weeks prior to the operative procedure. Ballantyne [8] also noted that hemorrhages which occur in patients with diabetes are extremely slow in absorbing. Besides the tendency to hemorrhage, the diabetic patient shows a greater incidence of postoperative infections. Thus, Gifford [9] reported on a group of young diabetic patients 43 per cent of whom lost eyes from infections following extraction of cataract. According to Reese,[10] however, since the use of insulin the tendency to infection among diabetic patients is no greater than among nondiabetic patients. There is also a greater liability to postoperative iritis and iridocyclitis in the diabetic patient, but in spite of all these dangers it is often necessary to operate on diabetic patients.

3. Gresser, E. B.: Am. J. Ophth. **14**:929, 1931.

4. Ballantyne, A. J.: (*a*) Tr. Ophth. Soc. U. Kingdom **52**:84, 1932; (*b*) **52**:86, 1932.

5. Hardy, W. F.: Am. J. Ophth. **5**:961, 1922.

6. Wheeler, J. M.: Tr. Am. Ophth. Soc. **14**:742, 1916.

7. Benedict, W. L.: Ohio State M. J. **21**:648, 1925.

8. Ballantyne.[4b]

9. Gifford, H.: Ophth. Rec. **20**:243, 1911.

10. Reese, F. M.: Internat. Clin. **3**:266, 1935.

The question of preparing the patient from the general standpoint should, of course, be left to a competent internist. It would seem from Benedict's experience that reduction of the sugar content of blood and urine to its minimum should be attempted by means of diet, but if the case is not controlled by these methods insulin should undoubtedly be used. However, the experience of my associates and me does not bear out that of Benedict's as to danger of postoperative hemorrhages after the use of insulin.

One cannot say that it is too dangerous to operate if the blood sugar content is over 200 mg. per hundred cubic centimeters and that it is safe to operate if the blood sugar content is under that amount, but the surgeon is driven almost by necessity to attempt surgical intervention if the patient is blind in both eyes and the condition is controlled as well as possible. In fact, Wilder [11] was so optimistic that he stated that complete control of the glycosuria is not essential and that the results of operation for cataract in the case of diabetic patients are as good as the results in the case of nondiabetic patients. This, however, is not the general opinion of operators of wide experience, who still feel that complications are more frequent in diabetic patients than in nondiabetic patients.

Hypertension as a complicating factor in cases of cataract cannot be ignored. Oláh [12] stated that even slight hypertension calls for venesection, with removal of from 200 to 250 cc. of blood on the day preceding the operation. Barrenechea [13] went even further and did a venesection on all patients over 50 years of age, unless there was some contraindication, removing from 250 to 300 cc. of blood. The reduction of the hypertension is, as a rule, the responsibility of the attending internist, and operative procedure is inaugurated only after he considers it safe. While it might be necessary to operate when the systolic pressure is above 200 mg. of mercury, the operation is done with a great deal of apprehension lest an intra-ocular or expulsive hemorrhage occur.

Nugent [14] in a large number of patients with cataract found the average systolic pressure to be 138, with a diastolic pressure of 80, and while, generally speaking, the patients with higher tensions were more likely to have complications, this was not necessarily so, since there seemed to be a greater number of complications in the group of patients with a systolic pressure of from 101 to 110 than in the group with a systolic pressure of from 141 to 150. While it seems hardly necessary

11. Wilder, R. M.: Symposium on Ocular Complications of Diabetes, Arch. Ophth. **12:**144 (July) 1934.

12. Oláh, Emil: Am. J. Ophth. **15:**626, 1932.

13. Barrenechea, A. S.: Arch. de oftal. hispano-am. **32:**207, 1932.

14. Nugent, O. B.: Tr. Sect. Ophth., A. M. A., 1933, p. 220.

to perform blood letting on every patient, as has been suggested, this is a procedure of merit in those cases in which the blood pressure is extremely high.

CHOICE OF OPERATION

Much has been written as to the operation to be selected for the different types of cataract, but there has been no absolute unanimity of opinion. Beach and McAdams [15] seem to be of the opinion that the intracapsular operation is applicable in cases of cataract of all types, while Knapp [16] agreed largely with Elschnig,[17] Kubik,[18] Wright [19] and others that the intracapsular operation is suitable in cases of senile cataract that is not in the intumescent stage and in cases of immature nuclear cataract. It is less suitable in cases of the morgagnian type and other hypermature types and in cases of cataract in the intumescent stage in which the capsule is so tense that grasping it is difficult. On the other hand, cataracts of the latter type can be extracted intracapsularly by using the eresiphake. The intracapsular type of operation is also considered unadapted in cases of cataract in patients under 50 years of age, because of the rather strong zonular fibers. Wright [20] decides against the intracapsular method in the presence of cough, asthma, high blood pressure, excitability and, in fact, practically any complicating general condition, as well as local rigidity of the pupil, high myopia, a fluid vitreous and glaucoma, and in the one-eyed patient. It would, therefore, seem that those who have had wide experience feel that extracapsular extraction is, on the whole, the safer procedure.

Choice of Operation for Congenital and Juvenile Cataracts.—In determining the type of operation to be performed on congenital cataracts, as well as on juvenile cataracts, the decision has to be made whether simple needling is to be performed or whether needling with subsequent lavage is to be practiced. The practice of needling combined with lavage after a few days is, in children, accompanied with such great danger of subsequent infection or injury from trauma that it seems to be rarely justified. The question of the time element is of little moment unless the child lives a considerable distance from a source of surgical help, so that the expense of travel becomes an important factor. What difference does it make to a child of 1 or 2 years if the period of absorption

15. Beach, S. J., and McAdams, W. R.: Intracapsular Extraction of Cataract in the Average Practice, Arch. Ophth. **15**:95 (Jan.) 1936.

16. Knapp, A.: Certain Aspects of the Intracapsular Extraction of Cataract by Forceps, Arch. Ophth. **16**:419 (Sept.) 1936.

17. Elschnig, E.: Die intrakapsulare Starextraktion, Berlin, Julius Springer, 1932.

18. Kubik, J.: Zentralbl. f. d. ges. Ophth. **32**:433, 1935.

19. Wright, R. E.: Am. J. Ophth. **16**:230, 1933.

20. Wright, R. E.: Am. J. Ophth. **20**:1, 1937.

is one month or six months? In a few cases the cataract may absorb with only one needling, although more than one is usually necessary, but a second operation is not necessary as long as the capsule is open and aqueous is coming in contact with the lenticular substance within the capsule. While Jackson [21] advocated the needling operation in patients up to 40 years of age, this is not the operation of choice unless slit lamp examination reveals that the lens is very soft and filled with large water clefts. In such cases the cataract may liquefy and absorb rather rapidly after the capsule is opened, but one should be ready at all times to open the anterior chamber and wash out the soft lenticular substance if secondary tension develops, which is not an unusual complication. Needling by the posterior route, which was recently advocated by Ballantyne [22] because of the lessened danger of swelling of the lens, does not seem to be well justified, since investigation has shown that the aqueous has the greater influence on the liquefying process which is the first essential to absorption.

ADVANTAGES AND DISADVANTAGES OF PRELIMINARY IRIDECTOMY

The question of the advisability of a preliminary iridectomy either as a routine measure or in cases of exceptional or complicated conditions, such as immature cataract, is one that has caused considerable discussion and called forth much difference of opinion. It is thought that von Graefe was the first to practice this procedure. Some operators since then have practiced it as a routine procedure, but a greater number employ it only in cases of more or less exceptional or unusual conditions. The chief advantages seem to be the following: first, the ripening effect on immature cataracts; second, the fact that at the time of the subsequent extraction there is no hemorrhage into the anterior chamber from the cut iris; third, the fact that one of the most painful parts of the operation does not occur at the time of the extraction, and, fourth, the fact that the surgeon becomes somewhat acquainted with the reaction of the patient to surgical procedure. Of the more or less doubtful advantages claimed by some might be mentioned the fact that there are an unobstructed region for insertion of the cystitome or capsule forceps, absence of iritis due to pressure and less need for a subsequent needling operation.

Of the disadvantages, the following seem to be of great moment: first, two penetrations of the ocular coats, with the accompanying dangers of exogenous infection; second, the increased expense of two hospitalizations, with the accompanying disturbance of home routine, and, third, the increased apprehension of the patient (it has been my experience that

21. Jackson, E.: Arch. Ophth. **42**:596, 1913.
22. Ballantyne, A. J.: Brit. J. Ophth. **20**:540, 1936.

when a patient undergoes a second operation he seems to dread it more than the first operation and complains of more pain than at the time of the initial operation).

In regard to the more recent methods of operating, the question of exogenous infection remains practically the same as in former times. The problem of lessening the pain is of less moment, for with the use of more prolonged application of the local anesthetic and especially with the use of retrobulbar injections, the pain caused by the operation is no longer an important factor. While there is undoubtedly a limited field for preliminary iridectomy, it is becoming progressively less important, especially as an operation to hasten ripening, since the intracapsular method of extraction is advocated for the immature cataract. My experience agrees with that of Fuchs, who stated that he rarely found indications for the operation.

POSTOPERATIVE COMPLICATIONS

Owing to the multiplicity of postoperative complications, it will be impossible to discuss all of them in a review of this scope, but the more frequent ones will be dealt with briefly.

Glaucoma.—Glaucoma following extraction of a cataract may result from the fact that the iris or the capsule of the lens has prolapsed and healed into the wound, which condition seems to disturb the normal drainage sufficiently to cause increased tension. On the other hand, there may be an ingrowth of epithelium into the anterior chamber, which, by completely epitheliating the filtration angle greatly or entirely, reduces filtration, with a resulting increase in intra-ocular pressure. I have observed two cases of this type in which the diagnosis was confirmed by microscopic studies, a third case in which a clinical diagnosis was made by means of the slit lamp and a fourth in which the condition was discovered in an enucleated eye. In the two cases in which the diagnosis was definite the eye was operated on by filtration methods, but even repeated operations failed to reduce the tension permanently. Vail [23] reported a case in which a clinical diagnosis of downgrowth of epithelium into the anterior chamber was made; the condition seemed to be benefited by radiation, but the diagnosis was not confirmed by microscopic investigation, so there is a possibility that this diagnosis was incorrect. In one of my cases, which has previously been mentioned, in which the eye was subsequently enucleated and the diagnosis was confirmed, the condition was treated by radiation, without any apparent beneficial result. If the increased tension is due to incarcerated iris or capsule, freeing the wound from the adherent capsule or iris will at times reduce the tension to normal. If the condition is due to epithelial

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23. Vail, D. T., Jr.: Tr. Am. Ophth. Soc. **33**:306, 1935.

ingrowth, a filtering operation should be attempted, as well as the use of radiation, but so far the results of such treatment have not been encouraging.

Endophthalmitis Phaco-Anaphylactica.—The rather pronounced reactions following extraction of cataract in a few cases, especially in those in which there is considerable soft lenticular substance, has been noted for many years. Verhoeff and Lemoine advocated the term endophthalmitis phaco-anaphylactica for this condition. A spirited discussion took place as to whether this condition is due to an allergic reaction, to some type of infection or even to the irritating properties of the lens itself. Gifford,[24] in this country, and Rötth,[25] as well as Riehm,[26] abroad, were on the negative side. Burky,[27] however, was able to produce a similar condition experimentally in rabbits by first sensitizing them to lens protein plus staphylococcus toxin, after which needling of the lens produced a marked ocular reaction. If the injections of the same combination were continued, the rabbit became desensitized, at which time needling of the lens produced little, if any, reaction. On the basis of these observations Burky and Henton[28] treated two patients whose condition was clinically diagnosed as endophthalmitis phaco-anaphylactica, with rapid and satisfactory improvement. It would therefore seem that if this complication arose, the combined treatment of lens protein with staphylococcus toxin was the method of handling such conditions.

Sympathetic Ophthalmia.—One of the most disappointing and disastrous complications to develop after extraction of cataract is sympathetic ophthalmia. Fortunately, this condition is infrequent, the incidence ranging from 0.027 to 0.5 per cent. However, if, in a case of bilateral cataract, after successful removal has been accomplished in one eye and after what seemed like a satisfactory extraction has been carried out on the opposite eye, it is found in the course of a few weeks that the eye previously operated on is inflamed and that there are photophobia, deposits on Descemet's membrane and all the other signs of sympathetic inflammation, the condition is almost certain to be sympathetic ophthalmia. Theobald[29] found that the condition occurred only twice in 7,444 cases in which extraction of cataract was performed, but in these cases the reaction was violent and ultimate vision very poor.

The question of treatment in cases of sympathetic ophthalmia is difficult because of the uncertain etiology. The use of large doses of

24. Gifford, S.: Tr. Sect. Ophth., A. M. A., 1925, p. 82.
25. Rötth, A.: Arch. Ophth. **55**:103, 1926.
26. Riehm, W.: Klin. Monatsbl. f. Augenh. **88**:62, 1932.
27. Burky, E.: Experimental Endophthalmitis Phaco-Anaphylactica in Rabbits. Arch. Ophth. **12**:536 (Oct.) 1934.
28. Burky, E. L., and Henton, H. C.: Am. J. Ophth. **19**:782, 1936.
29. Theobald, G.: Am. J. Ophth. **12**:597, 1930.

salicylates, as advocated by Gifford, has seemed, in my hands, to be as efficacious as any other treatment. Arsenicals given intravenously are sometimes useful, and Verhoeff [30] expressed the opinion that diphtheria antitoxin in large doses is effective. Desensitization to uveal pigment was recommended by Woods,[31] while Friedenwald [32] stated the opinion that ultraviolet irradiation of the general cutaneous surface is of benefit. Unfortunately, the sympathetic ophthalmia which occurs after surgical intervention for cataract seems to be violent, and treatment seems in most cases to be disappointing. ·

Postoperative Hemorrhage.—Postoperative hemorrhage into the anterior chamber is frequent, especially in diabetic patients, as was shown by Wheeler.[6] Vail [33] found, on averaging available figures, that it occurred in 7.59 per cent of all cases in which extraction of cataract was performed. Since it is more frequent in those cases in which iridectomy has been performed, it seems safe to assume that it frequently comes from the iris, but it is also seen occasionally in cases in which simple extraction has been done. The corneoconjunctival incision may be the source of the hemorrhage in a certain number of cases.

It has been suggested that the use of epinephrine at the time of operation was a factor in the production of the hyphemia, but Jensen,[34] who studied the question, came to the conclusion that there was no material difference in the frequency of bleeding in those cases in which epinephrine was used and in those in which it was not used. Schneider [35] noted a severe hemorrhage into the anterior chamber in a case of hemophilia, which caused a rupture of the wound that required suturing. The blood eventually absorbed, leaving an obstructing membrane. Prior to operation on the other eye, the patient was given intramuscular injections of calcium for three days before the operation, and a hypodermic injection of morphine hydrochloride and scopolamine hydrobromide one hour before the operation. This eye showed no bleeding after the operation, healed without complications and had good resulting vision. While hemorrhage into the anterior chamber is comparatively frequent and usually not serious, this cannot be said of expulsive hemorrhage, which is rare, fortunately (the incidence being 1 case in 1,000), but which practically always destroys the eye. This type of bleeding may occur at the conclusion of the incision or may be delayed as long as the tenth day after the operation.   .

30. Verhoeff, F.: Tr. Am. Ophth. Soc. **24**:173, 1926.

31. Woods, A.: Am. J. Ophth. **9**:52, 1926.

32. Friedenwald, J.: Am. J. Ophth. **17**:1008, 1934.

33. Vail, D. T., Jr.: Tr. Am. Ophth. Soc. **31**:496, 1933.

34. Jensen, V. A.: Acta ophth. **10**:382, 1932

35. Schneider, R.: Klin. Monatsbl. f. Augenh. **91**:365, 1933.

Most of the hemorrhages from the choroidal vessels which cause loss of the eye are not of the expulsive type that necessitate immediate enucleation but are often discovered at one of the dressings after the patient has complained of some restlessness and pain in the eye. Treatment of the milder conditions of hyphemia consist of the use of atropine and heat locally combined with the use of calcium internally. If repeated bleeding takes place, solution of brain extract may be given hypodermically, as recommended by Black,[36] who reported a case in which the hemorrhages ceased after its use. If the hemorrhage is one of the more severe types, injection of a preparation of ergot, solution of posterior pituitary (10 minims) and epinephrine hydrochloride (10 minims) given two or three times a day, as recommended by Ziegler,[37] may be used as a prophylactic measure, and may also be used if bleeding is actually taking place. Ziegler also advocated a hypodermic injection of morphine, hyoscine hydrobromide and pilocarpine hydrochloride. He stated that in spite of all treatment such eyes are usually lost. Thilliez,[38] on the other hand, claimed that the hemorrhage is immediately arrested after the subcutaneous injection of epinephrine hydrochloride and ergot. Although this opinion seems somewhat overenthusiastic, it behooves the surgeon to keep in mind these recommendations in case of an emergency. There seems to be a marked difference of opinion as to whether or not procaine hydrochloride and epinephrine hydrochloride, injected retrobulbarly, have a tendency to increase the number of cases of intraocular bleeding.

Inflammations, Purulent and Nonpurulent.—Infections of the eye following operations for cataract may be exogenous or endogenous, the former being the most frequent. Since greater care has been exercised in the preparation of the instruments and the conjunctival sac, as well as the patient, the incidence of postoperative infections has greatly diminished (from 5.47 to 1.5 per cent, according to Collins [39]).

The nonpurulent inflammations may be the result either of an infection or of trauma, the latter being often the cause. Ballantyne [8] advocated the use of atropine and hot compresses locally, with large doses of salicylates and sodium bicarbonate combined with mercurial inunctions. Foreign proteins are also helpful in many cases.

The treatment of the purulent infections is usually disappointing, but not every attempt is a failure; therefore, one should combat the

36. Black, M.: Am. J. Ophth. 7:539, 1924.

37. Ziegler, S. L., in Crisp, W. H., and Finnoff, W. C.: Contributions to Ophthalmic Science, Dedicated to Dr. Edward Jackson in Honor of His Seventieth Birthday by His Pupils and Colleagues in the United States, Menasha, Wis., George Banta Publishing Company, 1926, p. 7.

38. Thilliez: Bull. Soc. franç. d'opht. 35:406, 1922.

39. Collins, T.: Tr. Ophth. Soc. U. Kingdom 34:18, 1914.

condition as actively as possible. If one can obtain a culture showing the chief offending organism, a more logical treatment can be outlined. Prior to the identifying of the organism, the use of atropine with a local antiseptic such as a 20 per cent solution of mild protein silver or a 0.1 per cent solution of acriflavine, combined with the use of hot moist compresses applied almost continuously, is to be recommended. If the organism is a streptococcus, prontosil,[39a] given intramuscularly, may be tried. If the organism is a pneumococcus, ethylhydrocupreine hydrochloride in a 2 to 5 per cent solution may be helpful. The use of foreign protein in this condition often aids materially. Hartlieb [40] obtained good results by the intravenous use of 10 cc. of a 2 per cent solution of acriflavine hydrochloride and the intramuscular administration of 2 cc. of a mixture of partial antigens containing the metabolic products of various bacteria, lipoids from bile and neutral fats. Stargardt [41] expressed the belief that all soft tissue at the edges of the wound should be curetted away and the anterior chamber washed out with either physiologic solution of sodium chloride or a 1 : 5.000 solution of mercury oxycyanide. Butler [42] as well as Ellett,[43] advised cauterizing the margin of the wound with the actual cautery. If resolution does take place it is always a slow process.

Detachment of the Retina and Choroid.—Though detachment of the retina has been known since 1868, it has until recently been regarded as a rather infrequent complication in cases of extraction of cataract, although Fuchs [44] in 1900 found an incidence of 4.7 per cent. Recently O'Brien [45] stated that it occurred in 93 per cent of consecutive cases in which extraction of cataract was performed and in which the eye was examined immediately after the extraction. He reported that in this type of detachment the retina always becomes reattached, with no impairment of vision. Occasionally the detachment appears after several months and often in cases in which there is a history of a greater or less jar. In this group of patients there is usually the picture of typical detached retina, with loss of transparency. At times a rent is found in the detached portion, while frequently no such lesion can be detected. The prognosis is usually poor in these cases, but Cruise [46] reported

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39a. Prontosil is a derivative of sulfanilamide. The preparation used is said to be the disodium salt of 4'-sulfamidophenyl-2-azo-7-acetylamino-1-hydroxynaphthalene-3, 6-disulfonic acid.

40. Hartlieb, R.: Klin. Monatsbl. f. Augenh. **93**:526, 1934.

41. Stargardt, K.: Ztschr. f. Augenh. **43**:321, 1920.

42. Butler, T. Harrison: Tr. Ophth. Soc. U. Kingdom **40**:181, 1920.

43. Ellett, E.: J. Ophth., Otol. & Laryng. **10**:227, 1916.

44. Fuchs, E.: Arch. f. Ophth. **51**:199, 1900.

45. O'Brien, C. S.: Arch. Ophth. **16**:655 (Oct.) 1936.

46. Cruise, R.: Detachment Following Simple Extraction of Cataract, Arch. Ophth. **15**:576 (March) 1936.

successful results after the Šafář operation, and Hine [47] reported a cure by the same method in a case in which detachment occurred after the needling operation. Shapland [48] reported 30 cases of detachment following cataract, in 40 per cent of which holes were found. Of the patients who were subjected to operation, 18 per cent were cured. While the results in the hands of most operators have not been good in cases of this type of detachment, there is no doubt but that an attempt should be made to reattach the retina by one of the usual methods, the method employed being that which has seemed most successful in the hands of the attending surgeon.

Postoperative Delirium.—While postoperative delirium is a troublesome complication, it is usually not serious, since uncovering of the eye not operated on is usually all that is necessary to restore mental equilibrium. However, if the patient does not have a special nurse he may disturb the dressing or get out of bed and cause damage to the eye in the form of prolapse of the iris or an intra-ocular hemorrhage. Greenwood [49] found that the condition occurred in from 2.5 to 3 per cent of all cases in which extraction of cataract was performed. According to my own experience this seems somewhat too high, but possibly slight mental confusion might be present as frequently as Greenwood's figures indicate. McMullen [50] expressed the opinion that the mental distress which patients on whom extraction of cataract has been performed frequently have is due to anxiety as to whether they are going to see again. Bruns [51] allowed a group of patients on whom he operated to go home after the operation; he observed no mental disturbance in these cases, but it would seem that this treatment is slightly too heroic. The use of morphine and scopolamine or some of the milder hypnotics is usually all that is necessary to control the condition.

47. Hine, M. L.: Am. J. Ophth. **18**:558, 1935.

48. Shapland, C. D.: Am. J. Ophth. **17**:1159, 1934.

49. Greenwood, A.: Mental Disturbances Following Operations for Cataract, Tr. Sect. Ophth., A. M. A. 1928, p. 233.

50. McMullen, W. H.: Brit. J. Ophth. **20**:657, 1936.

51. Bruns, H. D.: Tr. Am. Ophth. Soc. **14**:473, 1916.

News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Publication of "Confinia neurologica."—A new international journal, to be called *Confinia neurologica*, is now being published by von S. Karger, A. G., Basel, Switzerland. The journal will be edited by Dr. E. Spiegel, of the Temple University School of Medicine. As far as the borderland of neurology is concerned, the new journal will endeavor to maintain the mutual relationship of neurology and surgery, otolaryngology and ophthalmology, syphilology, endocrinology and radiology. Complete and preliminary original articles, reviewing articles, society transactions and book reviews will be published. Articles may appear in English, French or German; the original papers will have a summary in all three languages. The summaries will be translated by the journal staff, if the author so desires.

SOCIETY NEWS

Eye Section of the Philadelphia County Medical Society.—The following papers were read at the scientific meeting of the Eye Section of the Philadelphia County Medical Society, held Oct. 7, 1937:

"Pathology of Sympathetic Ophthalmia," by Dr. Bernard Samuels, with discussion by Dr. Perce DeLong.

"Methods and Value of Examining Conjunctival Secretions," by Dr. Edgar B. Burchell, with discussion by Dr. Wilfred E. Fry.

Abstracts from Current Literature

Bacteriology and Serology

THEORETICAL BASIS FOR THE USE OF IMMUNOGEN B IN IMMUNIZATION OF THE EYE. G. G. BURSUK, Vestnik oftal. 10: 500, 1937.

Bursuk has been doing experimental work with antivirus since 1928. He observed that the products of activity of microbes accumulate in the antivirus and come in contact with the leukocytes which rush to the site of immunization. As a result of this interrelationship between the antivirus and the leukocytic ferments a new substance formed, which causes a sensitive reaction to the infection, i. e., rapid mobilization of the cellular elements and rapid formation of antibodies, the index of immunity of the tissues. This substance which appears during the process of immunization Bursuk named immunogen B. Through various experiments material accumulated which characterized the immunizing properties of immunogen B against the staphylococcus. The agglutination reaction was taken as the most accurate and simple. The same verified strain of Staphylococcus aureus has always been used for the agglutination. Bursuk also examined the change in the leukocyte count of the animals after the injection of immunogen B. Nine tables and one drawing demonstrate the results of the experimental study of immunization of the eye with immunogen B.

The following conclusions are drawn:

1. The immunogen against the staphylococcus causes a marked increase in the index of agglutination.

2. It also causes marked leukocytosis.

3. The increase in agglutinins and in leukocytes depends on the number of injections and the dose.

4. The action of immunogen B is specific, as it causes an increase in agglutinins for staphylococci only.

5. This was particularly marked in those animals the eyes of which were infected by staphylococci.

6. The concentration of the agglutinins and of the leukocytes was more marked near the site of the immunization; hence the first injection of the immunogen should be introduced at the site of the infection.

7. Retrobulbar injections are preferred, as they do not irritate the eye and cause increase in antibodies in the media; antibodies were prevalent in the aqueous of the posterior chamber twenty-four hours after the first injection of immunogen B, as compared with their number in blood vessels remote from the site of immunization.

O. SITCHEVSKA.

Conjunctiva

OCULAR PEMPHIGUS. W. J. KAPUSCINSKI, Ann. d'ocul. 174: 451
(July) 1937.

Ocular pemphigus usually follows pemphigus of the skin and usually
begins at the same time as pemphigus of other mucosa, particularly
buccal and pharyngeal. However it sometimes occurs as an apparently
primary ocular disorder.

Four cases of ocular pemphigus are detailed and are of interest
because of the complete clinical picture. Kapuscinski concludes that
ocular pemphigus is a disease characterized by primary degeneration of
the mucosa of the conjunctiva, especially the epithelium. The pathologic
processes in the cornea are limited to the superficial layer and may be
similar to the changes that take place in the conjunctiva. This chronic
inflammatory condition is characterized by cellular infiltration and is
purely a secondary phenomenon which is caused by the invasion by
microbes of tissue already pathologically altered by degeneration due to
pemphigus.

The article has fifteen illustrative figures and a bibliography.

S. H. McKEE.

SYPHILITIC CONJUNCTIVITIS. A. ŠPANIĆ, Arch. f. Ophth. 137: 312
(June) 1937.

Severe, subacute, unilateral, chiefly infiltrating conjunctivitis asso-
ciated with ipsilateral interstitial keratitis occurred in a syphilitic youth
18 years of age after the primary genital lesion. The conjunctivitis
resembled trachoma but remained unchanged under antitrachomatous
treatment. Pathologically, diffuse infiltration (by lymphocytes, plasma
cells and polymorphonuclears), with a few epithelioids and giant cells,
was seen. Under antisyphilitic treatment the disease subsided promptly.

P. C. KRONFELD.

ETIOLOGY OF GONOBLENNORRHEA NEONATORUM: REPORT OF A CASE.
E. JOHANSSON, Klin. Monatsbl. f. Augenh. 97: 775 (Dec.) 1936.

Three days after birth an infant had rhinitis, with a watery discharge,
which became purulent a few days later, when difficulty of nasal breath-
ing developed and traces of blood appeared in the nasal discharge. On
the fourteenth day the lids were slightly swollen, and a conjunctival
discharge was noted. Gonoblennorrhea neonatorum with chemosis was
evident in each eye on the sixteenth day. Gonococci were present in the
conjunctival and nasal discharge. Solution of silver nitrate had been
properly instilled after the method of Kredé. This case suggests the
necessity of considering not only diphtheria and syphilis, but also gonor-
rhea, as a cause of rhinitis in the new-born. Even stomatitis gonor-
rhoeica has been observed, due, probably, to gonorrheal conjunctivitis.
Johanssen suggests following Kredé's treatment with the use of less
irritating preparations in every case of maternal gonorrheal vaginitis,
so as to avoid late infections of the type described.

K. L. STOLL.

Cornea and Sclera

Cyclic Edema of the Cornea: Report of a Case. V. Haemmerli, Klin. Monatsbl. f. Augenh. **97**:745 (Dec.) 1936.

A laborer aged 19, employed at a quarry, had noticed in his ninth and tenth years that his sight was less acute in the morning than in the afternoon, and this condition had become worse in recent years; at first transient, it grew permanent finally. He never experienced headache, vomiting or epiphora, nor did he see colored halos around lights. The family history furnished no information as to ocular disease of the parents, sister and brother.

The patient's eyes were examined nearly every hour during the day and night; they appeared practically normal at 4 p. m. Early in the morning the entire cornea of each eye looked "finely dewed," probably as a result of fine epithelial changes. After 4 a. m. the fine "dewy" spots were more numerous and were scattered over all the layers of the cornea which now appeared thicker under the slit lamp. Incidentally, the intra-ocular tension was higher and fine "vacuoles" were visible in the epithelium of the cornea a few hours later. The vacuoles were less evident about 9 a. m. and had disappeared at about 11 a. m. A few green spots were caused by fluorescein while the vacuoles presented.

Despagnet and François described edema of the cornea unaccompanied by irritation or increased tension. Including Haemmerli's case, twelve cases of cyclic edema of the cornea have been described, the age of the patients ranging between 19 and 60 years. Haemmerli's patient was the youngest (aged 10 at the onset of the disturbance). The cycle occurred within twenty-four hours in every patient. Some authors considered hypercholesteremia, others, trophic disturbances, as the etiologic factor. The etiology is unsettled, in Haemmerli's opinion. Mydriatics, miotics, epinephrine and a cholesterol-poor diet have been used, and sclerectomy and iridectomy have been done, without result, by divers authors.

K. L. Stoll.

Corneal Transplants Taken from Cadavers: Report of Cases. Z. Nižetić, Klin. Monatsbl. f. Augenh. **97**:756 (Dec.) 1936.

Nižetić discusses divers technical questions relative to corneal transplantation and then reports his results in thirty-nine operations of this kind. In twenty-four of these cases the corneal transplants were taken from eyes of patients who had died of various causes, including enteritis, bronchopneumonia, asphyxia, intoxication with sodium hydrate, and accidents. The corneal disks were removed within from four to twenty-four hours after death. The ages of the deceased varied between 11 months and 30 years; eighteen of them were children. The technic is described. No enucleation was performed. No transplant was lost, but all of them healed, even two in cases in which infection occurred; these, however, grew opaque later on. Among these twenty-four cases, in five the transplant remained clear, in one case for over three years; in six it remained transparent, and in thirteen it became opaque. These results resemble those of other authors. Nižetić thinks that permanent transparency is less dependent on the material than on the substratum chosen for transplantation. The more the normal corneal elements are preserved in the substratum, the better will be the transparency of the

transplant. The cases were not especially selected with reference to fitness for a favorable keratoplasty, because the author's purpose was the study of the material; only a few of these eyes were free from complicated total leukoma.

<div align="right">K. L. Stoll.</div>

TWO CASES OF PERMANENTLY CLEAR HEALING CORNEAL TRANSPLANTS IN OPTICAL KERATOPLASTY. S. Ochi, Klin. Monatsbl. f. Augenh. **97**: 761 (Dec.) 1936.

Ochi reports two cases in which homoplastic, circumscribed, penetrating optical keratoplasty was performed. The patients were a woman aged 22 and a man of the same age. The transplants were taken from eyes enucleated for glioma in children who were 3 years and 4 years and 8 months old, respectively. Ochi selects for implantation the least vascularized portion of the cornea. He considers the transplants taken from children the best. The blood group of the patient and the donor need not be the same. The result, in Ochi's opinion, depends more on the corneal changes of the patient's eye than on the selection of the spot for the transplantation. It depends, secondly, on the age of the donor and finally on the technical experience of the operator.

<div align="right">K. L. Stoll.</div>

Experimental Pathology

IRITIS PRODUCED IN RABBITS' EYES BY THE INTRAVENOUS INJECTION OF CRUDE AND PURIFIED CULTURES OF BACTERIA ISOLATED FROM PATIENTS WITH CERTAIN INFLAMMATORY EYE DISEASES. C. BERENS, E. L. NILSON and G. H. CHAPMAN, Am. J. Ophth. **19**: 1060 (Dec.) 1936.

The authors give the following summary and conclusions:

"Iritis was produced in rabbits by the intravenous injection of either primary or purified cultures from 19 of 21 patients with acute or chronic eye diseases, and in 11 of 14 controls (laboratory assistants, healthy children and patients with arthritis and thyrotoxicosis).

"Positive results were obtained with various microörganisms as follows: streptococci (alpha, beta, and gamma types), staphylococci (albus and aureus), colon bacilli, nonlactose fermenters, enterococci, and Friedländer bacilli. Of the 51 primary cultures from patients with eye disease, 25.5 percent produced iritis in rabbits and 39 percent caused death of the rabbits before examination or too early for the production of eye symptoms. Of the 35 primary cultures from the control group, 26 percent produced iritis and 60 percent caused death of the rabbits before iritis was observed. The high mortality of the rabbits injected with primary nasal cultures accounts for the large number of undetermined results.

"Iritis was produced by 44 percent of 61 purified strains of streptococci from patients with eye disease as compared with 29 percent of 69 strains from persons in the control group.

"Of the other organisms from patients with eye disease, 36 percent of the 22 purified strains of staphylococci, members of the colon group, and enterococci produced iritis. The results were undetermined in 18

percent. In the control group, 41 percent of the strains of staphylococci, members of the colon group, and Friedländer bacilli produced iritis. The results were undetermined in 25 per cent.

"Of the total of 134 cultures from patients with eye disease, 36 percent produced iritis while 17.9 percent were undetermined. Of the total of 116 cultures from persons in the control group, 29.2 percent produced iritis in rabbits, while 21.5 percent were undetermined.

"Toxicity, as measured by in-vitro tests, did not seem to be related to the iritis-producing power of streptococci and staphylococci. Seventy percent of the organisms which produced iritis gave positive toxicity reactions, whereas 60 percent of the strains which did not produce iritis also gave positive toxicity reactions.

"It is concluded that, while iritis is produced in rabbit's eyes by various cultures of bacteria, this property is not characteristic of any one bacterial genus, neither is it distinctly a property of cultures from patients with inflammatory eye diseases."

<div align="right">W. S. Reese.</div>

General

The Degree and Prevalence of Vitamin A Deficiency in Adults. H. Jeghers, J. A. M. A. 109: 756 (Sept. 4) 1937.

The article deals with the study of one hundred and sixty-two students of the Boston University School of Medicine with especial regard to dark adaptation.

The author's summary and conclusions are as follows:

Vitamin A deficiency is common in adults and varies from a photometrically detectable phase to the complete clinical syndrome. In the group of medical students, 35 per cent had low photometer readings, and 12 per cent had clinical manifestations of the deficiency. The chief manifestations, in the order of their frequency, were night blindness, photophobia, dry skin, dry conjunctivae, blepharitis and follicular hyperkeratosis. The factors producing the deficiency were analyzed, and showed that the skipping of meals and poor choice of foods were chiefly responsible. After dietary analyses it was concluded that 4,000 U. S. P. units of vitamin A daily represents the minimal requirement for a healthy adult. Infections were more numerous and severe among the students with vitamin A deficiency than among the other students. Further evidence that it is dangerous for the hemeralope to drive an automobile at night was obtained. Photometric evidence of night blindness appeared in six days, and subjective evidence in five weeks, after the production of pure vitamin A deficiency in an experimental subject. Night blindness preceded gross epithelial changes.

<div align="right">W. Zentmayer.</div>

Ocular Headache. D. S. Stewart, Brit. M. J. 2: 59 (July 10) 1937.

Headache is a symptom of speculative etiology. A patient consults his physician only if the headache is persistent, recurrent or disabling. In cases of chronic headache it is as necessary to examine the optic disks as to test the urine, the blood pressure or the knee jerk. One should also determine if the patient can read 6/6 (that is, the seventh

line of the usual Snellen test type from a distance of 6 meters) with each eye separately. This test eliminates uncorrected but not overcorrected myopia.

Any severe illness, operation or accident, if it occurs in the late thirties or after, may leave the accommodation permanently lessened.

The normal relation between accommodation and convergence, and the imbalance between these two, which may occur in cases of uncorrected hyperopia are well known. In children the imbalance may lead to strabismus; in adults, asthenopia or headache may result.

Stewart thinks that the binocular balance at the reading distance is important in ruling out this group of conditions. He recommends the use of the Maddox convergiometer for discovering these disorders. Correction of refractive errors often restores the normal muscle balance for near.

In conclusion, he states that examination of the visual acuity, the optic disks and the lateral muscle balance at the reading distance should indicate which patients need ophthalmic attention.

W. F. DUGGAN.

OCULAR PAIN: ITS TREATMENT BY ANESTHESIA OF THE SPHENO-PALATINE GANGLION AND ORBITAL ALCOHOLIZATION. A. MAGITOT, Ann. d'ocul. 174: 361 (June) 1937.

Ocular pain brings the patient to the ophthalmologist as frequently as visual disturbances. Eliminating such causes as swelling of the lids, burns, and conditions caused by infection of a neighboring sinus or dental abscess, Magitot discusses only those related to the eyeball itself. Among these may be mentioned corneal erosions, which can usually be helped by the instillation of an analgesic.

Certain diseases of the anterior segment of the eye, such as kerato-conjunctivitis, scleritis, parenchymatous keratitis and iritis, are usually accompanied by considerable pain, which is treated by certain drugs, the choice of which varies for individual patients, and by the local application of hot fomentations. One may also try a method which is without risk and which sometimes gives remarkable relief; that is the subconjunctival injection of air. The technic is simple and consists in provoking subconjunctival emphysema. Magitot has been using this method since 1912 and injects the air at four cardinal points, obtaining a large swelling about the cornea. The emphysema is usually absorbed in about twenty-four hours but may remain a little longer. Sometimes these methods are insufficient, and it is necessary to use others. He suggests anesthesia of the sphenopalatine ganglion and injections of alcohol into the orbit. These methods are described in detail, and a commentary on the results is added.

S. H. McKEE.

General Diseases

FIBROCYSTIC DISEASE OF THE FRONTAL BONE (PAGET'S OSTEITIS). R. E. WRIGHT, Brit. J. Ophth. 21: 364 (July) 1937.

A Hindu girl aged 20 showed a soft cystic tumor projecting from beneath the left supra-orbital rim and proptosis of the left eye. A roentgengram revealed a "nigger wool" skull.

An incision was made into the cyst through the brow; a portion of the frontal bone was removed; the cyst was drained from the temporal side, and the skin was allowed to fall in. The periosteum at the orbital rim was continuous with the membranous wall or capsule of the cyst, which intervened between the orbital contents and the cyst cavity.

The histopathologic observations were consistent with the view that this was an instance of localized fibrocystic disease of bone falling within the group in which there is no change in the calcium and the phosphate metabolism.

The article is illustrated.

W. ZENTMAYER.

Glaucoma

RESULTS OF THE SURGERY OF GLAUCOMA. L. BOTHMAN and M. J. BLAESS, Am. J. Ophth. 19: 1072 (Dec.) 1936.

This article is an analysis of 100 cases (143 eyes) of primary glaucoma, including 7 in which there were retinal hemorrhages before operation and 8 (13 eyes) in which there was hydrophthalmos. No case in which there was evidence of iridocyclitis is included. The results of various operations on these eyes are discussed and tabulated. The authors give the following summary and conclusions:

"The anterior chamber was shallow and the cornea steamy when the tension was more than 50 mm. Hg.

"The 'middle pressure' was approximately the same as the intra-ocular tension in glaucomatous eyes under miotics and closely approximated that figure in patients with less than 50 mm. of pressure.

"Fields constricted to within a few degrees of the fixation point in glaucomatous eyes are no contraindication to surgical treatment.

"One late infection in fistulating operations occurred among 156 eyes and this one recovered 0.6 — 3 vision.

"The Elliot trephining operation was found to be the most satisfactory surgical procedure in chronic glaucoma."

W. S. REESE.

GLAUCOMA IN AMBLYOPIA. S. V. ABRAHAM, Am. J. Ophth. 19: 1094 (Dec.) 1936.

Abraham believes that glaucoma occurs infrequently in amblyopic eyes, as the blood supply is decreased in cases of subnormally functioning organs, and therefore these cannot call on the same extensive vascular response that is obtained in normal tissue. He believes that the reports in the literature bear out this hypothesis. He reports a case of acute glaucoma in an eye undoubtedly amblyopic following early convergent strabismus, and states the following conclusions:

"1. A case of acute glaucoma in an amblyopic eye is presented. 2. It is suggested that subnormally functioning eyes tend to be less susceptible to primary glaucoma. This seems to be true for amblyopic eyes. 3. Relief from 'eye strain,' especially as it occurs in hyperopia, is particularly desirable in glaucomatous patients. 4. The relation of a disturbed inflow to the etiology of glaucoma is emphasized."

W. S. REESE.

Lacrimal Apparatus

ACUTE DACRYOADENITIS DUE TO THE MORAX-AXENFELD DIPLOBACIL-
LUS. R. E. WRIGHT, R. BAHADUR and K. KOMAN NAYAR, Brit.
J. Ophth. **21**:367 (July) 1937.

A Hindu youth aged 19 had, in addition to inflammation of the right
lacrimal gland, enlargement and tenderness of the right preauricular
gland. Pulp from the preauricular gland yielded a pure culture of the
diplobacillus of Morax and Axenfeld.

W. ZENTMAYER.

LACRIMAL ELIMINATION OF DEXTROSE IN DIABETES. D. MICHAIL,
P. VANCEA and N. ZOLOG, Compt. rend. Soc. de biol. **125**: 194,
1937.

Normally the tears contain no detectable dextrose, but in the diabetic
patients examined the dextrose content ranged from 32 to 84 mg. per
hundred cubic centimeters. It is of interest that the lacrimal gland is
thus concerned in the elimination of products of abnormal metabolism.

J. E. LEBENSOHN.

DIVERTICULUM OF THE LACRIMAL SAC WITH SPECIAL REGARD TO
ROENTGENOLOGIC FINDINGS. F. SPINELLI, Arch. di ottal. **44**: 89
(Feb.) 1937.

The literature on the subject of diverticulum of the lacrimal sac is
reviewed, and a case of this condition is reported. The patient, a
woman of 31, had noticed a swelling over the right lacrimal sac since
the age of 8 years. It increased in size gradually, being the size of a
small nut when seen by Spinelli. It extended to the middle of the lower
lid and was soft. Pressure on the tumor caused no escape of pus
through the canaliculi, but a few drops of pus could be expressed by
pressure on the region of the lacrimal sac. Irrigation into the nose
was possible. Puncture of the cyst revealed pus containing diplococci.
Its contents were replaced by 20 per cent iodized oil. Roentgenograms
showed a large oval shadow without apparent connection with the sac.
Later roentgenograms, however, made after injecting the lacrimal sac
with iodized oil, showed that the cyst was filled from the sac through
a small communication.

The lacrimal sac and the cyst were removed. Sections showed the
cyst to be composed of tissue identical with that of the lacrimal sac,
a septum of similar tissue separating the two cavities. Apparently the
septum was arranged so as to form a valve which opened only on
pressure from the side of the lacrimal sac, in which respect the condition
seems to the author unique among those reported in the literature.

S. R. GIFFORD.

Lens

PNEUMATIC EXTRACTION OF THE LENS BY ZONULAR RUPTURE. H.
LAGRANGE, Ann. d'ocul. **174**: 387 (June) 1937.

Lagrange has manufactured a pneumatic extractor for the extraction
of cataract, which he wishes to bring before practitioners and which

substitutes for other methods rupture of the zonule by traction at its ciliary insertion.

The method of anesthesia is explained, and the necessity of care in following out the procedure is emphasized. The avoidance of haste is most necessary. The contraindications are also given.

The article does not lend itself to abstraction. There are five pages of illustrations showing the details of the method and the appliances used.

S. H. McKEE.

PHYSICOCHEMICAL INFLUENCES AFFECTING OPACIFICATION OF THE LENS. P. REISS, J. NORDMANN and C. REISS, Compt. rend. Soc. de biol. 125: 464, 1937.

To a filtrate of triturated lenses a small amount of an oxidizing agent, such as hydrogen peroxide, quinone or 2, 6-dichlorphenolindophenol, was added. Preserved by a drop of toluene, the mixture was then incubated at 37 C. for twenty-four hours. Only a slight clouding was noted in solutions of normal or alkaline p_H, but even a slight acidification, as at p_H 6.5, produced a marked effect. With the oxidation-reduction potential of the solution with the added hydrogen peroxide at $+ 368$ millivolts, opacity is manifest at p_H 6.9. Fresh lenses immersed in these solutions reacted in the same manner.

J. E. LEBENSOHN.

Neurology

OCULAR CHANGES IN MULTIPLE SCLEROSIS. D. MARSHALL and R. G. LAIRD, Am. J. Ophth. 19: 1085 (Dec.) 1936.

Marshall and Laird emphasize the frequency and early appearance of ocular changes in multiple sclerosis. They report a series of one hundred cases in which this condition was diagnosed by the department of neurology of the University of Michigan Medical School; 75 per cent of the patients had a routine examination by the department of ophthalmology. The ocular manifestations of multiple sclerosis are discussed, and a case of multiple sclerosis in which ocular changes were found is reported. The following summary is given:

"A review of history and ocular findings in 100 consecutive cases of multiple sclerosis is presented. In general the frequency of ocular pathology corresponds closely with statistics reported by previous writers. In addition a case of hemianopsia is reported in which diagnosis of multiple sclerosis was based entirely on field changes. The article aims to remind ophthalmologists and neurologists of the important and frequent part the eye plays in the diagnosis of the disease."

W. S. REESE.

SUPRASELLAR MENINGIOMA: REMOVAL IN TOTO, WITH PROMPT AND LASTING AMELIORATION OF VISION. P. PUECH, P. HALBRON and L. GUILLAUMAT, Bull. Soc. d'opht. de Paris, February 1937, p. 71.

A patient aged 28 years had rapid loss of vision, which decreased to 5/10 for the right eye and to 2/10 for the left eye. There were pale disks and bitemporal hemianopia. The results of neurologic exam-

ination were negative. There was slight involvement of the clinoid processes, although the sella turcica was normal. A diagnosis of suprasellar tumor was made. The visual fields are reproduced, and six drawings of the large tumor made during operation are included in the article. Differentiation from adenoma or craniopharyngioma was easily made. One month after operation the visual fields were normal, and vision of the right eye was 9/10 and that of the left eye 10/10. The tumor was removed in a single mass.

L. L. MAYER.

NYSTAGMUS IN TUMORS OF THORACIC PORTION OF SPINAL MEDULLA. H. HOFF and O. PÖTZL, Med. Klin. 33: 598 (April 30) 1937.

Hoff and Pötzl observed spontaneous nystagmus in three of six patients with tumors in the thoracic region of the spinal medulla. They admit that because of the small number of cases the figures are of no significance, but they think that the characteristics of the nystagmus which appeared in these cases deserve attention. They observed that it had a slow and a rapid component. The rapid component was in the direction of the visual movements; it was entirely horizontal and appeared especially when the patient was standing, sitting or in the abdominal or the dorsal position. Turning the pelvis or rotating the legs arrested the nystagmus. These seemed to be the only movements that caused its cessation. To be sure, in one case completion of the lateral recumbent position was necessary to arrest it. After surgical removal of the tumor from the thoracic portion of the spinal medulla, the nystagmus disappeared completely, or only slight traces of it remained. As long as the nystagmus was at the peak of its development pelvic movements toward either side and each lateral recumbent position had the same effect, in spite of the fact that in the first two cases the spastic and paretic conditions were not equally severe on the two sides. In evaluating the significance of the described observations the authors point out that these indicate the need of careful examination of the nystagmus that develops in cases of tumor of the spinal cord. Even if spontaneous nystagmus is absent, it is advisable to subject the patients to the same examination as that carried out on patients with spontaneous nystagmus in order to determine whether some change in the position might not perhaps elicit latent nystagmus. The significance of this type of nystagmus is slight in regard to the differential diagnosis, because it occurs also in rare cases of multiple sclerosis; but it should be impressed on the nonspecialist in this field that the existence of nystagmus does not necessarily speak either against a tumor of the thoracic portion of the spinal cord or for multiple sclerosis.

J. A. M. A. (W. ZENTMAYER.)

Ocular Muscles

LESIONS OF THE OCULAR MUSCLES BY DIRECT TRAUMA. L. VERICO, Arch. di ottal. 44: 65 (Feb.) 1937.

Two cases of lesion of an ocular muscle or of ocular muscles caused by trauma are reported. In the first case a wound was caused by a piece

of iron, which tore the bulbar conjunctiva, severed the internal rectus muscle near its insertion and partially severed the reflected portion of the superior oblique muscle. Operative repair was performed immediately. The severed portion of the internal rectus muscle with Tenon's capsule was united to the stump of the tendon, and portions of the tendon of the superior oblique muscle which presented in the wound were removed. Fairly good motility was obtained, but a divergence of 10 degrees was left, for which tenotomy of the external rectus muscle was performed only two weeks later. The eye became straight in the primary position, and there was slight limitation of motion only in the lower inner field.

The second patient received a severe laceration of the lids by falling on the handle of a door. The laceration exposed the lower half of the globe and severed the inferior rectus muscle at the junction of its tendinous and muscular portions. Immediate suture was performed. The muscle was found by grasping deeply into the orbit and determining whether the muscle tissue was held by the forceps by traction produced on attempted movements of the eye. The tissue was sutured to the stump of the tendon, and an excellent result was obtained. After a month motility was practically normal, and no diplopia was present.

The mechanism of injuries to the ocular muscles and the relative value of early and of late operative intervention for such injuries are discussed. A bibliography is given.

S. R. GIFFORD.

Ocular Muscles

INFLUENCE OF LUMBAR PUNCTURE ON THE PARALYSIS OR PARESIS OF THE EXTRINSIC OCULAR MUSCLES. F. CARAMAZZA, Rev. oto-neuro-oftal. 12: 529 (Nov.-Dec.) 1936.

Caramazza describes nine cases of paralysis of ocular muscles of differing etiology. There was no increase in the intracranial pressure. In six cases improvement followed lumbar puncture. Results can be obtained only when the paralysis is of recent origin. On the basis of his observations the author considers the subject worthy of study by ophthalmologists.

G. BONACCOLTO.

Operations

TARSORRHAPHIA MEDIALIS VERA. N. I. SHIMKIN, Brit. J. Ophth. 21: 343 (July) 1937.

The author's summary in part supplies an adequate abstract of the article.

Shimkin offers a new method of operation, tarsorrhaphia medialis vera, for ectropion paralyticum of the lower lid in cases in which the punctum lacrimale and the canaliculus lacrimalis are so occluded that the physiologic function of the lower lacrimal passage cannot be restored.

The operation consists in drawing the inner part of the lower lid, extending from the punctum lacrimale up to the inner canthus, by means of two loop sutures upward and inside a deep pocket in the inner part of the upper lid; the pocket is made by splitting the margin of the upper lid along the intermarginal line into two plates, extending from the punctum lacrimale superior up to the ligamentum internum.

The punctum and canaliculus of the upper lid remain intact in this operation.

On the medial part of the lower lid the skin is previously excised, and the conjunctiva and margin of the lid are scarified, which causes adhesion, when healing, to the tissues of the walls of the pocket in the upper lid. All the three layers are fastened together with a single knot suture in the newly formed canthus of the eye and with a loop stitch inward from it.

The operation is called tarsorrhaphia medialis vera, as the tarsus of each lid is sutured in the inner canthus of the eye, and not only the skin of the lids, as in all the methods of tarsorrhaphia medialis devised up until now. This operation makes it possible for the surgeon to gage the necessary distance to raise and shorten the lower lid. The cosmetic result is satisfactory.

W. Zentmayer.

Anesthesia Induced by Sodium Evipal in Ocular Surgery. Z. Nizetic, Ann. d'ocul. **174:** 375 (June) 1937.

The choice of an anesthetic depends not always on the physician, but often on the patient who says he will have the operation only if he is "put to sleep first." This is usually true of operations on the globe.

Nizetic describes the use of evipal ($C = C = $ cyclohexenyl $= N = $ methylbarbituric acid) and gives a table of data for 115 cases in which this anesthetic was used, showing the sex and age of the patients, the kind of operation, the amount of drug used and the results. He summarizes the findings as follows:

Narcosis induced by sodium evipal is, without doubt, like that induced by other narcotics, a serious matter, but the author has not had the slightest accident or serious complication from the use of this drug, probably because he kept the dose moderate. The results have been most satisfactory in ocular surgery, in which this form of anesthesia has such particular advantages as allowing freedom of the operative field, improvement in securing asepsis, and absence of preoperative and postoperative inconveniences. Sodium evipal is a satisfactory drug for inducing anesthesia and may be used in urgent ocular procedures and in major operations, and the length of anesthesia is sufficient for all ocular operations (?—S. H. M.). In operation for detachment of the retina Nizetic believes sodium evipal to be the anesthetic of choice.

S. H. McKee.

Orbit, Eyeball and Accessory Sinuses

The Inflammatory Pseudotumors of the Orbit (Birch-Hirschfeld). A. Franceschetti and E. Rutishauser, Arch. f. Ophth. **137:** 93 (April) 1937.

The term inflammatory pseudotumor of the orbit (Birch-Hirschfeld) designates a number of diseases which clinically cannot be distinguished from neoplasms and which only on pathologic examination prove to be inflammatory in nature. In this paper four cases of such a pseudotumor are reported; in three of the cases the process had started in the lacrimal gland, and in the fourth case it originated from the

periapical granuloma of a premolar. The pathologic picture was that of diffuse chronic inflammation in the case of the growth presumably of dental origin. In the other three cases the pseudotumor consisted chiefly of follicles in which, in two of the three cases, tubercle bacilli could be demonstrated histologically. In one of these two cases the bacilli proved to be pathogenic for the guinea-pig. In view of the relative frequency of pseudotumor the authors recommend that a biopsy be carried out early in all cases of suspected orbital tumor. Partial excision of a pseudotumor often leads to complete cure but may also cause considerable limitation of the movements of the eye and lids. The authors therefore favor simple incision instead of partial excision. "The formation of lymph follicles is a typical reaction of the orbital tissues."

P. C. KRONFELD.

CEPHALOCELE ORBITAE ANTERIOR. A. MIKLÓS, Arch. f. Ophth. 137: 222 (June) 1937.

Miklós reports two cases of anterior cephalocele of the orbit (one case is that of a patient operated on by Kreiker and reported by him in 1922). Exploratory puncture is recommended to establish the diagnosis. Early operation is indicated unless the cephalocele is accompanied by more serious developmental defects or by hydrocephalus.

P. C. KRONFELD.

Physiology

AN INVESTIGATION INTO THE THEORIES ON THE FORMATION AND EXIT OF THE INTRA-OCULAR FLUIDS. J. D. ROBERTSON, Brit. J. Ophth. 21: 401 (Aug.) 1937.

This study is considered under the following heads: historical, experimental, discussion, conclusions and summary. The paper occupies forty-five pages of the issue. Under the heading discussion there is an exhaustive analysis of the experimental work of other investigators. Robertson's conclusions and summary are as follows:

Conclusions.—The views put forward in favor of dialysis for the production of the intra-ocular fluids have been carefully investigated, and it is suggested that the evidence is unconvincing.

There is ample evidence to show that in the production of the aqueous there must be an expenditure of energy in the posterior chamber, for the membrane through which fluid passes into the chamber to become aqueous has an irreversible permeability.

There is evidence to show that there is no chemical equilibrium between the blood and the aqueous. Easily diffusible constituents, such as urea, sugar and uric acid, are not present in equal concentrations in blood as compared with the corresponding aqueous.

There is evidence that a physical equilibrium does not exist between the blood and the aqueous and that the equilibrium level of the intra-ocular pressure is not maintained by the hydrostatic force in the capillaries minus the difference in the osmotic pressure between the aqueous and the blood.

It is suggested that the evidence, chemical, hydrostatic and osmotic, points to the formation of the aqueous by a process of secretion, and

in addition there is evidence of the necessity for expenditure of energy in its formation.

Summary.—It is held that the formation of aqueous takes place by a process of secretion at the ciliary body and that this fluid moves forward to the angle of the anterior chamber and is actively absorbed into the canal of Schlemm by some process other than osmosis.

W. Zentmayer.

Further Research on Respiration of Ocular Tissues. D. Michail and P. Vancea, Compt. rend. Soc. de biol. **125:** 185, 1937.

Experiments both in vitro and in vivo indicate that epinephrine, insulin, cevitamic acid and the roentgen rays stimulate the oxidizing activity of lenticular and corneal tissue and tissue of the lacrimal gland. This investigation was suggested by a study of naphthalene cataract, in which the oxidizing power of the lens was found to be reduced from the onset of intoxication, preceding the retinal and lenticular lesions by several days and reaching its maximum with complete cataractous development.

J. E. Lebensohn.

Hydrogen Ion Concentration of the Tears. A. Gardilčić, Arch. f. Ophth. **137:** 71 (April) 1937.

In a case of cicatricial ectropion with exposure of parts of the upper fornix, the spontaneously formed lacrimal fluid was aspirated directly from the outlets of the lacrimal gland. The rate at which fluid appeared on the conjunctival surface is not given in the paper. The entire conjunctiva was in a state of chronic inflammation. The p_H of the undiluted lacrimal fluid (determined with a quinhydrone electrode and a capillary electrometer) was found to vary between 7.53 and 7.95. Dilution with double-distilled water lowered the p_H. The secretion of the conjunctiva is more alkaline than the tears.

P. C. Kronfeld.

Retina and Optic Nerve

Aneurysm of the Internal Carotid Artery with Atrophy and Compression of the Optic Nerve. J. O. Wetzel, Am. J. Ophth. **19:** 1053 (Dec.) 1936.

Wetzel briefly reviews the literature on aneurysm of the internal carotid artery producing changes in the optic nerve due to pressure. He reports a case of this disorder in a 39 year old housewife who showed a secondary atrophy of the optic nerve of the left eye, which progressed. The patient was observed for one year, at the end of which she died suddenly. Autopsy revealed extensive hemorrhage of the brain and a much flattened and stretched left optic nerve over the surface of an aneurysm of the left internal carotid artery. Other changes were observed in the body. The author discusses the etiology of aneurysm.

W. S. Reese.

MARKED AMELIORATION OF TABETIC OPTIC ATROPHY BY TREATMENT WITH AN ARSENIC AND BISMUTH PREPARATION. H. LAGRANGE and P. LEFÈVRE, Bull. Soc. d'opht. de Paris, February 1937, p. 58.

Lagrange was prompted by the report of Bégué, who found atrophy of the optic nerve following the use of acetarsone, to ask the specialist in syphilis, Lefèvre, to report on a certain patient. The patient had paralysis of the third nerve, associated with ptosis and diplopia. Treatment with mercurials injected intravenously resulted in cure of these paralyses. Prolonged treatment with intramuscular injections of acetarsone in doses of 1 Gm! resulted in decreased visual acuity after the third injection. Visual acuity was reduced from 9/10 for the right eye and from 10/10 for the left eye to 7/10 for each eye. Acetarsone was discontinued. Bismuth and mercury preparations were given, but vision decreased, and atrophy of the optic nerve was noted. The reduction of vision was to 2/10 for the right eye and to 1/50 for the left eye. A preparation of arsenic and bismuth in combination was then given. Visual acuity increased to 7/10 for the right eye and to 3/10 for the left eye. The authors feel that a trivalent arsenical is indicated in cases in which atrophy of the optic nerve is present.

L. L. MAYER.

PAPILLITIS AND STELLATE RETINITIS OF THE RIGHT EYE; DISCUSSION OF THE ETIOLOGY. DUBOIS-POULSEN and R. ROSSANO, Bull. Soc. d'opht. de Paris, February 1937, p. 99.

In the examination of a woman aged 36 the authors noted papillitis of the right eye and reduction of vision to 1/10; on second examination, five days later, a star figure in the macula, was seen. A thorough and painstaking general examination failed to reveal the cause of the condition. There was no evidence of kidney disease, or of hypertension, either local or general. This condition has not infrequently been reported in the literature (Bull, Eales, Leber, Larsson, Moore, Miles, Miley, Webster and Schlesinger having described cases) as evidence of focal infection of some distant organ. The tests used in the present case are reported in detail. In addition to the question of the diseases previously mentioned, the authors discuss that of toxic disease, disease of blood cells, trauma, syphilis and tuberculosis. Therapy directed specifically toward these disease processes was of no avail in the authors' case. It is necessary to conclude that the etiologic factor is unknown, and the authors ask for further discussion to reveal the cause of such a syndrome.

L. L. MAYER.

CEREBRAL ANGIOSPASM: SIGNIFICANCE OF RETINAL ARTERIAL HYPERTENSION. F. SCHOUSBOÉ, Bull. Soc. d'opht. de Paris, February 1937, p. 112.

From a study of fifty-three patients with clinical characteristics of cerebral lesions of angiospastic origin the following conclusions may be drawn:

1. Forty-three per cent of the patients had no modification of the retinal circulation (local spasm).

2. Fifty-seven per cent had accompanying retinal arterial hypertension.

In 79 per cent of the latter the condition was transitory; in 34 per cent of this group spasm of retinal arteries was bilateral, and in 66 per cent the condition was on the same side as the cerebral lesion. In thirty-three patients the etiologic factors could be determined, and these were grouped as follows:

Posttraumatic epilepsy.................	1
Essential hypertension..................	12
Syphilitic hypertension................	5
Hypertension with endogenous intoxication (urea)	8
Hypertension with exogenous intoxication (tobacco or alcohol)................	3
Raynaud's disease.....................	1
Reflex angiospasm.....................	3

L. L. Mayer.

New Anatomic Findings in Choked Disk. C. Behr, Arch. f. Ophth. **137:** 1 (April) 1937.

Behr condemns alcohol-hardened specimens of choked disk and the conclusions based on such specimens, on the grounds that the rapid dehydration of the edematous nerve is bound to lead to the formation of holes in the tissues which were not present intra vitam. For seven years he has, therefore, embedded his specimens of choked disk in gelatin. Sections prepared in this manner reveal no gaps between the bundles of nerve fibers and the septums, no empty spaces under the pia and no "loosening" of the axial strand. The essential pathologic change visible in these gelatin sections is edema of certain portions of the nerve fiber substance itself. In cross-sections this edema manifests itself as multiple but minute empty spaces within the bundles of fibers. The multiplicity of these spaces gives the affected portion a cystic appearance. In early stages of choked disk only the subpial bundles show this appearance, but with the progress and the longer duration of the disease bundles of fibers situated more axially become affected. The extent of the pathologic process parallels the loss of the visual field. In longitudinal sections the bundles of fibers show swelling except at the places of contact with the septums. Thus a "rosary" shape of the bundles results. The septums and the axial strand appear compressed and not edematous. The entire extracranial portion of the optic nerve shows this change. The swelling is confined to the disk and does not involve the adjoining retina.

Ophthalmoscopically, choked disk starts as a circumscribed swelling of one quadrant of the disk. According to Behr, this fact can be explained only by his own theory, namely, that of the existence of a centripetal intrafascicular lymph stream in the optic nerve, which is interfered with by increased intracranial pressure. Only live nerve fiber tissue is capable of undergoing the edematous change which underlies the "choking," another fact which is most easily explained by Behr's theory. The hydrops of the intervaginal spaces cannot exert pressure on the nerve because, according to Behr, these spaces communicate with the orbit.

P. C. Kronfeld.

Vitreous and Retinal Detachment. K. Lindner, Arch. f. Ophth. **137**: 157 (June) 1937.

The paper deals chiefly with the theory of Leber, Gonin and Lindner that retinal tears are caused by the pulling effect of an abnormal vitreous. Leber formulated his views as follows: A disease of the peripheral choroid causes shrinking and, consequently, detachment of the vitreous. Progression of this shrinking process puts the retina under tension and produces the retinal tear. Gonin's addition to this theory was the assumption that the tear occurs at the place where previous retinochoroidal disease has produced an adhesion between the retina and the vitreous. Such adhesions often persist throughout life without doing any harm. After a minor trauma which entails jarring of the vitreous, the retina tears at the place of the adhesion. Lindner's contribution was chiefly a study of the mechanics of these traumas. He states: "Only movements of the eye around its center of rotation are dangerous." The inertia of the detached and freely movable vitreous constitutes the pulling factor. In exceptional cases tears are caused by small portions of vitreous which are adherent to the retina while the bulk of the vitreous has become liquefied.

The paper contains chiefly histologic evidence in favor of Gonin's theory. The pathologic diagnosis of detachment of the vitreous is sometimes difficult. The presence of a subvitreal space filled with coagulated fluid is a reliable sign. If the subvitreal space is empty, pathologic changes of the detached hyaloid membrane or precipitates on the inner surface of the retina (thickening, breaks) indicate the presence of vitreous detachment in vivo. Using these criteria, Lindner diagnosed pathologically vitreous detachment in a large number of eyes with retinal detachment. Gonin's theory postulates that an eye with retinal detachment shows the following pathologic picture: (1) the vitreous is detached; (2) the detached vitreous is connected with the peripheral (anterior) edge of the tear, and (3) the posterior edge of the tear is free, and the subvitreal space communicates through the tear with the subretinal space. In a large number of eyes Lindner has found that these requirements were fulfilled. This is the most common mechanism of the formation of a tear, the entire mass of the still solid vitreous pulling on a relatively small retinal area. Other possible factors in the formation of a tear are the pulling effect of a small portion of vitreous adherent to the retina but unattached to the rest of the vitreous (which in these cases is liquefied) and a degenerative process in the retina without any pulling action of the vitreous. For the study of the relations between the vitreous and the retina Lindner recommends fixation after the method of Szent Györgyi (*Ztschr. f. Mikr.*, vol. 31, p. 23).

P. C. Kronfeld.

Ophthalmoscopic Diagnosis of Choked Disk. F. Schieck, Arch. f. Ophth. **137**: 203 (June) 1937.

In early stages of choked disk Schieck has observed delicate exudates within the vascular funnel which during the progression of the disease spread forward and onto the surface of the disk. There they cause bulging of the internal limiting membrane into the vitreous. During the

subsidence of the choking the limiting membrane returns relatively late to its normal position. Exudates of this form do not occur in optic neuritis or in pseudoneuritis. Gullstrand's reflex-free ophthalmoscope is less suitable for the detection of these fine exudates than is the ordinary hand ophthalmoscope. The best method of examination, of course, is that of focal illumination (ophthalmoscopy in focal light, slit lamp microscopy of the fundus).

<div align="right">P. C. KRONFELD.</div>

HOMOLOGOUS CHANGES OF THE RETINAL PIGMENT EPITHELIUM AND OF THE EPITHELIUM OF THE RENAL TUBULES IN VARIOUS FORMS OF POISONING. Y. KOYANAGI and C. KINUKAWA, Arch. f. Ophth 137: 261 (June) 1937.

The inorganic iodine preparation septojod has been known to produce, on intravenous injection, acute degeneration of the retinal pigment epithelium in man and the rabbit. Koyanagi and Kinukawa have found that septojod also affects the kidney of the rabbit, causing albuminuria, oliguria and the appearance of epithelial casts and leukocytes in the urine. Pathologically, the distal portions of the tubules are principally affected. The epithelium of the tubules contains hyaline droplets, and the cells undergo various other degenerative changes and form hyaline cylinders in the lumens, a picture which fits into that of nephrosis. Similar changes in the kidney are produced by naphthaline. The renal poisons mercury bichloride, uranium salts and guaiacol, on the other hand, cause, on injection into the carotid artery, alterations of the retinal pigment epithelium, which the authors interpret as defensive reactions, with subsequent degeneration. In all these forms of poisoning the authors consider the retinal changes and the renal changes to be homologous. Koyanagi reports the clinical and pathologic observations in one case of eclampsia. The renal observations he interprets as being typical of tubular nephrosis, while the retina only showed "secretory activity" of the pigment epithelium. "The small arteries of the choroid showed in places pronounced hyalin, the lumen obliterating degeneration." The outer retinal layers (except the pigment epithelium) were normal, a fact which, according to the authors, makes the explanation of the lesion of the pigment epithelium on a vascular basis difficult. Koyanagi and Kinukawa consider these lesions to be of toxic origin. Contrary to the reports of other authors, they found that in cases of typical albuminuric retinitis the pigment epithelium exhibits changes which are analogous to those produced in the rabbit by the aforementioned poisons. In the opinion of Koyanagi and Kinukawa, albuminuric retinitis and the homologous tubular changes are caused by toxins and not by angiospasm.

<div align="right">P. C. KRONFELD.</div>

Tumors

PAGET'S DISEASE OF THE EYELID ASSOCIATED WITH CARCINOMA. A. HAGEDOORN, Brit. J. Ophth. 21: 234 (May) 1937.

Paget's disease (in some features resembling Bowen's disease) of the eyelid was observed histologically in the eyelids removed from a patient with trachoma and xerosis. One year later the appearance of a

subcutaneous nodule necessitated exenteration of the orbit. On histologic examination this nodule proved to be a carcinoma simplex.

Histologic examination of the lid showed changes strongly resembling certain varieties of basal cell carcinoma.

The article is illustrated.

W. ZENTMAYER.

ORBITOPALPEBRAL RHABDOMYOSARCOMA. MONBRUN and G. OFFRET, Bull. Soc. d'opht. de Paris, February 1937, p. 78.

Muscular tumor is rare and one of the orbitopalpebral junction is an exception. The literature contains few reports of muscular tumor about the eye. In 1924 Redslob reported three cases of such a tumor. Mayer, Lopez-Henriquez and Cardenas Pupo found tumor of the lids. Mier reported a case of rhabdomyoma of the orbit. Monbrun and Offret removed a mass from the right palpebro-orbital junction in a child 9½ years old. The tumor grew so rapidly that the palpebral opening was greatly enlarged. Application of radium was of no avail in stopping the progress of the growth. A photograph of the child shows the tumor, and a drawing demonstrates the histologic picture of the removed tissue. The results of macroscopic and of microscopic examination are outlined in detail. Four types of cells were noted: (1) fusiform cells, which were greatly increased in number; (2) young muscle fibers, which were seen in great abundance and were very long; (3) a double striation of ordinary muscle fibers, and (4) abnormal cells—round cells with chromatin of various coloration. The authors conclude that the tumor had its origin in the superior oblique muscle.

L. L. MAYER.

Vitreous

EXPANSION PRESSURE OF VITREOUS DEPRIVED OF ITS SALTS. J. GOEDBLOED, Arch. f. Ophth. 137: 127 (April) 1937.

The volume of vitreous deprived of its salts by immersion in distilled water increases up to 200 per cent of the original value. During this gradual loss of salts and increase in volume a low pressure due to expansion can be shown to prevail, which decreases at approximately the same rate as the volume increases.

P. C. KRONFELD.

Sympathetic Ophthalmia

PROPHYLAXIS OF SYMPATHETIC OPHTHALMIA. B. NAKAMURA and Y. UCHIDA, Arch. f. Ophth. 137: 233 (June) 1937.

Two hundred and thirty-four patients with conditions which fulfilled the requirements of (1) perforating injury with subsequent iridocyclitis of the injured eye, (2) injury to the ciliary body, (3) sympathetic irritation of the uninjured eye and (4) sensitiveness to tuberculin were divided into two groups. The first group received prophylactic treatment consisting of injections of tuberculin (the Japanese tuberculin designated as tuberculin AO. one injection of 1 cc. a week being given) and injections of calcium iodide. In this group no patient showed

sympathetic ophthalmia, while in the second group of patients, who received no such prophylactic treatment, nine of the one hundred and thirty-nine subjects showed sympathetic ophthalmia. It is impossible to tell from the data given whether or not the two groups were—except for the prophylactic injections—treated alike, that is, whether or not the number of enucleations which might be considered as a prophylactic measure was the same in both groups. The authors are convinced of the prophylactic effect of the tuberculin treatment.

<div align="right">P. C. KRONFELD.</div>

Therapeutics

CLINICAL OBSERVATIONS ON TREATMENT OF PURULENT DISEASES OF THE EYE WITH IMMUNOGEN B AGAINST THE STAPHYLOCOCCUS. G. G. BURSUK, E. I. ZAKRJEWSKAYA, B. A. SCHULTZ and K. A. HELLE, Vestnik oftal. **10**: 522, 1937.

The action of immunogen B against the staphylococcus was observed in 661 hospital patients. Of these, 137 had corneal ulcer, 90 purulent inflammation of the uveal tract, 4 dacryocystitis, 3 phlegmon of the orbit and 7 blepharitis, and for 420 the treatment was applied as a prophylactic measure.

The immunogen was given intramuscularly every other day; the dose was from 3 to 6 cc., and up to six injections were given. A general reaction was usually present; an increase in temperature, general malaise and an increase in the leukocyte count were observed in 68 per cent. There were treated: ulcers due to the staphylococcus (fifty-eight cases), ulcers due to the pneumococcus (twenty-nine cases) and corneal ulcers of mixed origin (fifty-one cases).

The most favorable results were obtained in the cases of the ulcers due to the staphylococcus; the hypopion absorbed on the fifth or the sixth day, and epithelization of the cornea occurred on the tenth to the eleventh day, with fair improvement of vision.

Treatment with milk and diphtheria antitoxin was tried for the purpose of comparison. The average length of hospitalization of the patients treated with the immunogen equaled fourteen and a half days, while patients treated with the other foreign proteins spent about twenty-six days in the hospital.

Immunogen B proved to give favorable results in traumatic iritis, while in traumatic uveitis and endophthalmitis the inflammatory process was not influenced by the treatment.

In all the cases of severe blepharitis (seven) the treatment with immunogen B gave good results. As a prophylactic measure the immunogen was injected into all patients who had to undergo an intra-ocular operation and also into all those with perforating injury of the eye. In nearly all the cases the course was an aseptic one.

The article is extensive and contains nine tables illustrating in detail the clinical data.

<div align="right">O. SITCHEVSKA.</div>

Society Transactions

GERMAN OPHTHALMOLOGICAL SOCIETY

Fifty-First Annual Meeting, Heidelberg, July 6-8, 1936

TRANSLATED BY PERCY FRIDENBERG, M.D., New York

DOCENT DR. M. BÜCKLERS, *Tübingen, Reporter*

Third Scientific Session

Tuesday, July 7, 8:30 a. m.

DR. LINDBERG, Helsinki, Finland, *Chairman*

VII. INSULIN AND THE EYE. DR. REINHARD BRAUN, Rostock.

The reader presented data based on clinical observations of 700 diabetic patients in the metabolism department of Umber in the Berlin-Westend Hospital, especially in regard to the experience with insulin in its ophthalmic therapeutic relations. He never observed any ocular damage. Even patients who had had frequent and severe insulin reactions and whose blood sugar content accordingly fluctuated markedly showed no retinal changes of any kind. Retinitis diabetica of patients under treatment with insulin runs a course which is no more hemorrhagic than is that of patients with other conditions. It is true that treatment with insulin does not seem to help the retinal disease process. On the other hand, the reader noted in 2 cases the disappearance of diabetic opacities of the lens, and in 2 other cases similar cataractous forms remained stationary for years. Insulin is of the greatest value for diabetic patients with senile cataract; since its general introduction and use the risk in operation for extraction is no greater for these patients than for patients with normal metabolism. There did not seem to be any greater tendency to hemorrhage than in the patients not under treatment with insulin. The reader was not able to get any definite idea of the relation of insulin to the intra-ocular tension. A definite observation showed, on the other hand, that hypotony of the globe is not necessarily and invariably present in diabetic coma. The usual lowering of the intra-ocular tension in this terminal condition may possibly have to be attributed to the contrast or opposition of the insulin-producing apparatus and the chromaffin system. In a case in which drops of pilocarpine had been given by mistake to a patient who was under treatment with insulin given in large doses the reader observed hypoglycemic reactions. The diabetogenic nervous diseases of the eye have evidently become very rare. In a series of diabetic patients even careful perimetric examination failed to reveal any contraction of the visual field or any scotoma. The improved treatment of diabetic patients and the use of insulin may have something to do with this. This applies also to the diabetic oculomotor disturbances which have manifestly become much less frequent than of yore.

DISCUSSION OF PAPERS VI AND VII

Dr. H. Gasteiger, Frankfort-on-Main: I was able to study a series of cases of diabetic retinitis in which the blood pressure was normal, beyond a doubt. The cases were controlled and observed as to diet, constitutional treatment and so on by the Volhard clinic in Frankfort, and the blood pressure was invariably found to be normal. So one must agree with Dr. Mylius that increased blood pressure is not to be considered the decisive etiologic factor in diabetic retinitis. It was further advised that manometric measurements of the pressure in the central retinal artery be instituted, which might perhaps add to the knowledge of the origin of the ocular lesions.

Dr. Bartels, Dortmund: On the basis of my experience with extensive clinical material, I feel that the question of insulin and a possible casual relation to ocular hemorrhages has not been definitely cleared up. In 2 women with cataract, kept under observation for years before operation, hemorrhages were never found. As preliminary treatment to extraction of cataract the patients received 20 units of insulin three times daily. The patients, who had had diabetes for years, had never been subjected to treatment with insulin. After a perfectly smooth intracapsular extraction of a cataract one of the patients suffered severe retinal hemorrhages and the other, massive hemorrhages into the vitreous, which abolished sight at once, at least for a time. In the course of the next few weeks, with an insulin-free diet, the hemorrhages absorbed, and full sight was regained. In these cases it is quite possible that insulin had something to do with the hemorrhages.

Dr. Gilbert, Hamburg: I had a similar experience. After the most careful preliminary treatment with insulin there was a severe hemorrhage in the eye first operated on, only the smallest part of which cleared up. Thereupon the treatment with insulin was stopped and the extraction operation on the other eye put off. When this operation was performed, after two months, the much feared complication did not materialize. This observation fits in rather well with what Dr. Bartels has told us.

Dr. Caanitz, Kiel: In a man aged 30, in the course of treatment which rapidly made his urine free from sugar, there developed, in addition to a transitory increase of hypermetropia amounting to 2 D., an isolated total paralysis of accommodation which came on suddenly, overnight, and disappeared completely in three weeks.

Dr. A. Pillat, Graz, Austria: In a study of 600 patients with diabetes who had been repeatedly subjected to a complete ophthalmologic examination, the number of ocular complications was found to be over 60 per cent, and I am inclined to attach rather more importance to the increased intra-ocular tension as an etiologic factor in the causation of retinal lesions. It is to be noted specially that in the overwhelming majority of patients with changes in the fundus observation with the Gullstrand ophthalmoscope shows retinal edema in the region of the macula or/and along smaller and larger vessels and that this edema is often present previous to the appearance of hemorrhages or white foci. In regard to damage due to insulin, long-continued use of this drug

seems without a doubt to favor the occurrence of hemorrhages in the retina and also that of pathologic lesions in the vessels of the choriocapillaris. Cataract in diabetic patients can be operated on, after suitable preparatory internal treatment, with the same confidence as ordinary senile cataract. Repeated tonometric examination showed, in an appreciable percentage of cases, increased intra-ocular tension without any clinical signs of glaucoma, if only the blood pressure was high.

Dr. M. Bücklers, Tübingen: Dr. Braun has expressed the opinion that insulin can arrest incipient opacities of the lens in diabetic patients. The following clinical observation would seem to speak against this: The case is that of a young girl with severe diabetes (the blood sugar content was over 300 mg. per hundred cubic centimeters) who died within a year. In spite of the continued administration of large doses of insulin, typical diabetic cataract developed, first in one eye and then in the other, with the characteristic high content of fluid. The fundus showed no changes in the nature of diabetic retinitis.

Dr. W. Meisner, Cologne: Has any of my colleagues who have observed so many diabetic patients seen the very serious forms of glaucoma, first described by Salus, in which there is a marked, diffuse, hyperemia which is uniformly distributed over the iris?

Dr. C. Mylius, Hamburg: In answer to Dr. Gasteiger, the pressure in the retinal vessels was not taken. Attacks of glaucoma like those mentioned by Dr. Meisner were not observed. In answer to Dr. Pillat: There was certainly no retinal edema in the early cases, and there was no increase in the blood pressure.

Dr. Reinhard Braun, Rostock: I agree with Dr. Mylius as to the tendency to retinal edema in diabetic eyes. I have examined many eyes with the Gullstrand ophthalmoscope and could not convince myself of the presence of any edema in diabetic retinitis. I am in the fortunate position of being able to contribute some data as to the question of the etiology of diabetic retinitis, although these point in a different direction from those already presented, on the basis of 2 clinical observations.

Two patients aged 23 and 26, respectively, presented the fundus picture of typical diabetic retinitis. Both showed changes in the blood chemistry which may have given some support to a theory of the etiology. In one case the residual nitrogen content of the blood had increased to 56.8 mg. per hundred cubic centimeters, in the other to 44 mg. Both patients had a perfectly normal blood pressure. In the 23 year old woman there was clinically also a condition of renal change which probably was to be interpreted as a chronic nephritis, as the urine showed albumin, erythrocytes and casts. In the other patient the renal finding was clinically normal. Dr. Pillat's suggestion to guard against a hypoglycemic reaction by having the patient fast for a day preceding operation seems dangerous, as it is just in hunger conditions that the tendency to hypoglycemic states, i. e., insulin shock, seems most marked. In regard to the case reported by Dr. Bücklers, the question arises as to how the patient had been treated. Under Umber's influence I have come to the point of insisting on strenuous overinsulinization in cases of diabetic cataract. Whether or not, and to what extent, this was carried out in the case reported by Dr. Bücklers I cannot decide.

VIII. Familial Angiopathy in Juvenile Retinal Hemorrhages and Hemorrhages Into the Vitreous. Dr. O. Marchesani, Munich.

In the sibs of patients with adolescent intra-ocular angiopathy (periphlebitis, hemorrhages into the vitreous, retinitis proliferans) there is not infrequently found a cumulative occurrence of vascular lesions of the most varied sort. In 5 sibs, reported on in detail, the following diseases were observed: gangrene of the extremities, cerebral paralyses, ulcus ventriculi, varicose ulcers of the legs, apoplectic attacks, rheumatism and vasoneuroses.

These are the same pathologic processes which are so often to be found in adolescent persons with intra-ocular hemorrhages and which had previously induced the reader to consider the retinal disease as a manifestation of Buerger's thrombo-angiitis obliterans. Iridocyclitis, too, and rheumatism are rather frequent in cases of retinal periphlebitis and in the kinship of the patients. This observation is significant, as Rössle has lately included thrombo-angiitis obliterans in the general category of rheumatism, and many new points of view present themselves for the interpretation, *inter alia,* of the etiology of uveitis. One of the specially characteristic signs of the familial angiopathy under consideration is the occurrence of capillary lesions which are similar to those seen in severe vascular neuroses, and which, besides, are generally associated with capillary hemorrhages. The familial occurrence of the circulatory disturbances, which are to be considered as pathologically and pathogeneticaly uniform, speaks in favor of a hereditary anlage. Examination of the patients or a study of their history shows that some have had infections of one sort or another or have been subjected to toxic influences which may well have played a rôle in determining the disease. In others it is impossible to find evidence of any such irritative pathologic factors (*Reize*). The hereditary anlage seems to play the principal part. This anlage may, of course, probably be inherited in a greater or lesser quantity and/or intensity and, in a given case, suffice in the course of life to lead to manifestation of the disease, of its own account. The result of this research represents an important contribution to the question of the etiology of thrombo-angiitis obliterans.

<div align="center">DISCUSSION</div>

Dr. Engelking, Heidelberg: The family trees presented by Dr. Marchesani have not convinced me. If one wishes to prove any such causal relations it is quite unpermissible to include in the clinical picture as related to thrombo-angiitis obliterans, offhand, any and every case of rheumatism and all sorts of other diseases, such as ulcers of the lower portion of the legs or open sores on the feet, without an exact characterization of these conditions. In establishing a genealogy only those cases should be used, primarily, which allow the disease picture one is looking for to be recognized in a truly typical form. In the last few years I have taken great pains in cases of retinal periphlebitis to detect signs of Buerger's disease. Although I had the cooperation of internists and other specialists, I have not so far found a single case of this condition. Nevertheless, I would not go so far as to claim (deny ?—P. H. F.) that the clinical picture of retinal periphlebitis is *also seen in* cases of

Buerger's disease. It must, however, be expressly stated with emphasis that periphlebitis of the retina is also a definite manifestation of tuberculosis and, as such, is frequent. I myself have records of many cases which, in this regard, leave no room for doubt.

Dr. W. WEGNER, Freiburg: The family trees presented by Dr. Marchesani appear to me to prove very little. On the contrary, I feel that Dr. Marchesani has fallen into the opposite error to that which was generally made formerly, when every condition was considered as due to tuberculosis. He tries to relate everything to Buerger's disease, the essential nature of which is still obscure and insufficiently explained. I surveyed material consisting of from 65 to 70 cases of typical retinal periphlebitis. Among these cases, on careful analysis, just 2 were found which could most probably be considered as cases of Buerger's disease. Among the others, there was 1 case of severe pulmonary disease with formation of cavities; 1 female patient died of tuberculous meningitis, and a number of other patients had serious pulmonary complications which were unquestionably tuberculous. Hence I believe that the idea of a causal relation with tuberculosis is the most natural and obvious one, although one certainly cannot consider retinal periphlebitis in all cases as tuberculous.

Dr. M. BÜCKLERS, Tübingen: At the last meeting of Bavarian ophthalmologists I presented a clinical study of 10 patients with retinal periphlebitis who had been examined with great care for the symptoms described by Dr. Marchesani as characteristic of retinal angiopathy (Buerger). In the meantime the number of patients has increased to 16, without any unquestionable cases of Buerger's disease having been found. On the other hand, 7 of 8 patients had a positive tuberculous (Meinicke) reaction. Vasoneuroses of medium severity occur comparatively often in Würtemberg. Some of the lesions in other organs reported by Dr. Marchesani are so striking that they could not possibly have been overlooked. Dr. Marchesani may be able to tell how many of Buerger's numerous patients had periphlebitis retinae. So far I cannot help having the impression, considering the small family trees adduced, that in these cases chance correlations were mistaken for system diseases, i. e., pathologically related processes.

Dr. FLEISCHER, Erlangen: The etiology of retinal periphlebitis and of relapsing hemorrhages into the vitreous of adolescent persons has not as yet been made clear by any means. On the other hand, Dr. Marchesani's observations are important, and it behooves all of us to institute intensive investigation in the direction pointed out by him. Still it is more than questionable whether obliterating thrombo-angiitis (Buerger) is a simple clinical and pathologic entity, specific and sharply defined, and whether it is not, perhaps, the expression of various pathologic factors. Among the latter one must certainly reckon tuberculosis. In the case first described by me the anatomicohistologic observations were those of severe pulmonary tuberculosis, to which the patient finally succumbed. In this case, then, the assumption of tuberculosis of the eye as well, and especially of the retina, seems very probable.

Dr. W. ROHRSCHNEIDER, Cologne: What is the geographic distribution of cases of thrombo-angiitis obliterans in Germany? In Cologne, too, all patients with retinal periphlebitis are systematically and carefully

examined for evidence of this underlying systemic disease. So far the results have invariably been negative. What is more, internists and neurologists agree that although they are familiar with the original works of Dr. Marchesani and his collaborators, they have not been able to determine the presence of thrombo-angiitis obliterans in the population of Cologne.

Dr. Cause: An observation which shows in the most striking way the familial relation of this vascular disease is that in the case of a student aged 24 in whom the juvenile type of retinal hemorrhages had developed seven years previously. Five months later there occurred severe apoplexy, leading to left hemiplegia, which even today makes itself felt in a very disturbing manner. In the subsequent years retinal hemorrhages occurred frequently, but there was no recurrence of any serious cerebral lesion. The patient was examined repeatedly and thoroughly last year in the Cologne clinic, and later in that of Volhard, but investigation in every direction failed to reveal anything beyond a hemorrhagic diathesis. The patient's father died at the age of 48, of periarteritis nodosa. His was the unusual case of a rare disease, in which the diagnosis was made during life in the course of an exploratory abdominal operation. The disease had dragged on for months with severe but indefinite symptoms, when acute cholecystitis developed, requiring extirpation. Even during the operation the discoloration and pallor of the large vessels had been remarked; this appearance was so striking that they could hardly be distinguished from the common bile duct. So there was undoubtedly the inheritance, at least, of a tendency to hemorrhagic diathesis in the case of the son. As a factual correction, the case had been under continuous observation for seven years, during which time the typical lesions of retinal periphlebitis, and hemorrhages of all varieties and in all layers and regions of the retina, as well as preretinal effusions of blood, had been observed.

Dr. A. Pillat, Graz, Austria: To facilitate thorough and intelligent discussion of the question which Dr. Marchesani has so happily broached, I make the suggestion that the name thrombo-angiitis obliterans be given to the condition which he considers to be retinal angiopathy. The term retinal periphlebitis, with the addition of "tuberculosa," should be applied to that condition in which, often under one's very eyes, white striations appear along the retinal blood vessels, and in which typical nodules form, generally at the bifurcations. Certainly, in view of the clinical observations and histologic research already presented, the occurrence of true tuberculous periphlebitis retinae cannot be denied. A sharp differentiation of these two diseases, including that of nomenclature, will facilitate future discussion.

Dr. C. Mylius, Hamburg: The etiology of periphlebitis is not identical in all cases. As to the factors principally discussed here, tuberculosis and Buerger's disease, further proof will have to be furnished. For the etiology of tuberculosis in many cases, the following observation has almost the value, and is practically the equivalent, of an experiment. The case is that of a young law student, who had been treated at Kiel since 1927 for typical unilateral periphlebitis with recurring hemorrhages. At first no consititutional disorder was found. Later on the roentgen rays showed pulmonary tuberculosis. The patient

spent half a year in a sanatorium, and the process in the lungs improved temporarily. However, fresh intra-ocular hemorrhages occurred repeatedly, almost every fortnight, it was said, toward the end of 1934. Early in 1935 the clinical picture was that of typical retinal periphlebitis with numerous hemorrhages. Reye, then in Hamburg, made a diagnosis of tuberculous tonsillitis. The tonsils, which were removed, were proved to be tuberculous by histologic examination and experimentation on animals. Since the tonsillectomy there have been no more intra-ocular hemorrhages. In June 1936 the patient had completely recovered, with full vision and the fundus picture of retinitis proliferans.

Dr. O. Marchesani, Munich: Even if the genealogical charts were not convincing on account of an apparent or claimed discrepancy in the nature of the clinical pictures adduced, in numerous cases the picture is unquestionably and unequivocally pathognomonic. In a series of these cases the disorder leads to the milder symptom complexes which are characterized predominantly by the microscopic capillary observations. Rössle's pathologico-anatomic studies give reason to believe that certain forms of rheumatism are to be included in this category. Although instances of retinal angiopathy accompanied by definitely proved systemic tuberculosis are adduced, one must appeal to the general experience, which is that this observation is extremely rare in comparison with the rather frequent occurrence of my symptom complex. In Buerger's disease, retinal angiopathy has been noted by me and by other observers repeatedly. The geographic distribution of thrombo-angiitis obliterans is probably not uniform, as the disease is more frequent in the East. If it is rarely diagnosed in certain regions, the reason is probably that the clinical picture of thrombo-angiitis obliterans, in the broader sense in which it is conceived by me, is but little known as yet. Without going so far as to say that tuberculosis has no significance in the etiology of this disease, it may be stated that tuberculosis is no more important than other unspecific infections and other pathologic factors. The vascular lesions in the interior of the eye are not typically or characteristically tuberculous; they have the same appearance whether tuberculosis or some other pathologic factor has been the determining cause. I feel myself justified in this conception by my histologic studies of the eyes. All the specimens I could possibly lay hands on were submitted again, quite lately, to a succession of expert pathologists, and all agreed that the sections were not tuberculous.

IX. Clinical Course and Pathogenesis of Detachment of the Vitreous. Dr. A. Pillat, Graz, Austria.

The occurrence of this lesion in the living human eye must be considered as proved. From the anatomic point of view, i. e., according to the location of the detachment of the vitreous, one may distinguish between (1) posterior detachment and (2) anterior detachment. The first includes all detachments of the posterior portion of the limiting layer of the vitreous from the ora serrata to its attachment at the optic papilla, including the ring-shaped tearing away from the latter. Anterior detachment is accordingly the breaking loose or recession retraction of the much less extensive portion of the limiting layer of the vitreous which lies in front of the base of the vitreous. Posterior detachment

of the vitreous may be divided into five subordinate groups, viz.: (*a*) superior detachment with the subdivisions funnel-shaped and tentlike, respectively; (*b*) posterior detachment *sensu strictiori*, i. e., that which occurs in the posterior third of the globe, with its three subdivisions, cul-de-sac-like detachment, ring-shaped breaking away from the papilla and peripapillary detachment without this break: (*c*) total posterior detachment, in which usually the greater part of the mass of the vitreous sinks downward and forward (A. Vogt); but in the living eye one rarely sees what is common, probably as an artefact, in the gross anatomic specimen, viz., the entire vitreous lying directly back of the lens; (*d*) lateral detachment, which rarely comes under observation, and (*e*) inferior detachment. Misleading pictures may appear on slit lamp examination, owing to various conditions. Thus, parts of the vitreous may undergo condensation and acquire a "membranous appearance," simulating a limiting membrane, or, on the other hand, fluidification may cause the development of large, optically empty, cystic cavities. Pathogenetically one may distinguish an inflammatory and a noninflammatory (senile-degenerative) form of this lesion. The first form includes those conditions in which an inflammatory transudate or exudate in the choroid or the retina lifts up the posterior portion of the vitreous membrane from the retina, as well as those in which inflammatory products in the vitreous itself, such as white blood cells, fibrin, blood and so on, become active by bringing about a decrease of volume due to alkalinization or acidification. The noninflammatory, degenerative form is seen in myopia, in retinitis pigmentosa, in cases of detachment of the retina, in apparently sound senile eyes and after intracapsular extraction of cataract. The pathogenesis of this form can be clearly understood only if one takes into consideration three factors, the absence of which (1 and 2) or the presence of which (3) assures the normal form of the vitreous: (1) the senile shrinking of the base of the vitreous, combined with disintegration of structure in the mass of the vitreous itself; (2) senile dehydration, and (3) loosening the normal attachment of the vitreous to the retina in old age. However, the senile changes in the chemistry, form and structure of the vitreous are not based exclusively on processes in this tissue alone but depend on conditions in the choroid and retina, some of which, at least, ophthalmologists have not learned to recognize or to interpret clinically and ophthalmoscopically. Detachment of the vitreous constitutes a hazard only when pathologic adhesions have formed between the vitreous and the retina.

X. The Various Forms of Detachment of the Vitreous as Observed with the Angulated (Gewinkelten) Microscope of Lindner. Dr. H. Rieger, Vienna, Austria.

The data for 350 cases of detachment of the vitreous which originated in various ways are reported and analyzed. Referring to numerous illustrations, some diagrammatic and others drawn in color from life, the reader discussed first the three forms of detachment of the vitreous which he distinguished, viz., superior, partial posterior, and complete posterior (see also the report of the transactions of the Ophthalmological Society in Vienna on Jan. 20, 1936, in *Ztschr. f. Augenh.* 89: 247, 1936). He then took up in detail the structure and

composition of the opacities located in the detached posterior portion of the limiting membrane. Most of the dense opacities which were formerly characterized as vitreous opacities and assigned a place in the tissue of the vitreous itself lie in the region of the detached posterior portion of the limiting membrane. These opacities have a characteristic, individual appearance which is determined largely by the age of the patient, the basic pathogenetic factor of the detachment and the region of the fundus which corresponds to the site of the opacity. The cases of detachment of the vitreous are to be divided into those in which there are holes in the region of the opacity and those in which there are no such holes. In the latter variety one is probably dealing with a simultaneous detachment of that delicate membrane composed of glia fiber cells which Salzmann has named the central glia-meniscus and which lines the walls and covers the floor of the excavation and also, although to a lesser degree, the surface of the disk itself. In juvenile subjects the limiting membrane detaches very easily, so that detachment without the formation of holes is comparatively frequent in young patients, although a similar form is seen quite often in senile subjects. In the juvenile form there is rarely an extensive dense opacity in the detached limiting membrane, whereas the senile, as well as the inflammatory, variety is characterized by dense opacities of definite form. In these cases it is probable that thickening or densification has taken place in the prepapillary glia-cuticulum.

Variations in the degree of such changes in the tissue account for the multiplicity of pictures of the opacities, increased as it is by the fact that these opacities, especially in old age, often show the formation of holes. Opacities located in the limiting membrane can also probably develop as a result of thickening of the hyaloid membrane caused by pathologic processes—mostly inflammatory—in the neighborhood of the disk, in the macular region or in the periphery of the fundus. These opacities are generally characterized by a multiplicity of holes. In conclusion, the reader discussed the significance of the condition of the detached vitreous itself for certain definite details of the clinical picture of this lesion.

DISCUSSION OF PAPERS IX AND X

Dr. W. Comberg, Rostock: My illustration of a detachment of the vitreous which showed itself in the region immediately behind the lens was published in 1924 (*Klin. Monatsbl. f. Augenh.* 72: 695, 1924). The topographic relations could be seen and interpreted exactly with the slit lamp. The vitreous was thickened. The posterior surface of the lens lay quite free in its upper portion, bordering on a space filled with fluid and bounded posteriorly and below by the vitreous. I have maintained the conviction for some time that there is a physiologic prolapse (*Durchhängen*) of the vitreous, either of its structural framework alone, including the remains of the hyaloid artery, or, what is more probable, of the colloidal vitreous besides. Observers who use the slit lamp know that the remnants of the hyaloid artery run backward and downward, obliquely, from its point of attachment to the posterior surface of the lens. All other "contours" which are visible in the vitreous behind the lens follow the same oblique course. This uniformity of course and direction can be explained only by assuming that the regions

in question have gradually altered their position, sinking down in response to gravity in the course of life, as a result of man's upright position, and that the vitreous too sags downward (*durchhängt*). On the basis of these observations it is extremely interesting to note that most detachments of the vitreous take place above.

In view of this physiologic sag of the vitreous there must naturally result, in the course of life, a slight pull *e vacuo* which may lead to a detachment of the vitreous.

DR. K. LINDNER, Vienna, Austria: I have observed a number of cases in which the vitreous was detached to within a point close behind the lens. To Dr. Pillat's categories of degenerative and inflammatory detachment of the vitreous must be added that group in which the detachment is caused by fistulization of the globe. In cases in which this process or condition lasts for any considerable length of time the vitreous contracts, and this contraction is not fully compensated when the eye is closed, so that a detachment of the vitreous remains. Furthermore, it is not only the tongue-shaped or island-shaped attachments of the vitreous to the retina which cause retinal tears, but much more often those posterior detachments of the vitreous which show at the margin a protrusion of their edges toward the rear. Tongue-shaped islands of adhesion in cases of detachment of the vitreous cause a tearing out, with a cover to the hole *(Lochdeckel)*, which can then be found adherent to the detached vitreous, as already described with accuracy by Gonin. In case of an irregular margin at the detachment of the vitreous so-called flap tears *(Lappenrisse)* develop, the flap remaining attached to the detached portion of the vitreous. In almost every case of retinal detachment one finds a detachment of the vitreous as one of the preceding, partial, predisposing pathogenic factors. The retinal lesions, for the greater part degenerative, must then occur as the exciting factor at the point on which the vitreous exerts traction, either as an island or in the form of an irregular protrusion. Finally, it is the elastic shock of the vitreous which causes, in the first instance, a hole-shaped tear and, in the second, a flap-shaped rip *(Einriss)* of the retina. I formerly shared the opinion that retinal detachment could take place spontaneously in the presence of fluidification of the vitreous as a result of impact and traction on degenerated portions of the retina by the residual shreds and clumps *(Ballen)* of vitreous. This is, however, not the case, or at least very rarely so, as individual masses of vitreous have little kinetic energy or force and, besides, run out toward the optic papilla to a flat base, as the shock effect of the mass of vitreous in motion spreads and becomes distributed. A primary retinal tear can ensue only when this shock action is concentrated on a sharply limited area, such as the island-shaped adhesion which in cases of detachment of the vitreous persists behind the general margin of the detachment, or, again, on a marginal zone of adhesion which runs out toward the disk. It was not by any means theoretical considerations, but rather a careful and thorough sampling and analysis of all the specimens having a bearing on this point which brought these facts to my special attention and induced me, in view of the generally meager results of the interpretation of histologic observations in changes in the vitreous, to look for a method of detecting the presence of a detachment of the vitreous in the living subject as well. There is such a method today.

On the basis of this ophthalmologists now know that the great majority of spontaneous retinal detachments take place only in eyes which have already shown a detachment of the vitreous, which corroborates part of Leber's original theory. However, we are not yet able to recognize those cases in which the eye is especially jeopardized owing to the presence of a detachment of the vitreous, as the apparatus for examination now at our disposal does not enable us to inspect completely the boundaries of a detachment of the vitreous.

Dr. K. Velhagen, Halle-on-Saale: I have been interested for some time in the senile retinal atrophy mentioned by Dr. Pillat, a condition which has been described histologically more often and more thoroughly than ophthalmoscopically. According to my observations, this lesion lies predominantly, not extremely peripherally but in a somewhat intermediate position, in a ring. This is probably the limit of the region supplied by the arteria centralis retinae. Farther toward the periphery the blood supply comes from the ciliary circulation. The nature of the intermediate region, which is particularly exposed to atrophy, probably explains the relative infrequency of tears at the ora serrata and the frequency of tears in the intermediate zone. The observation of detachment of the vitreous after removal of the lens in its capsule reminds me that mention has been made repeatedly, in the Spanish literature, that retinal detachment too is decidedly more frequent after intracapsular than after extracapsular extraction of cataract.

Dr. A. Pillat, Graz, Austria: I feel that the condition illustrated by the sketch shown by Dr. Comberg might correspond to an anterior detachment of the vitreous, a condition which I have never seen in my clinical material. I agree with Dr. Velhagen that the senile changes in the retina are located principally in the region of the equator or at least are most marked in that area of the fundus. A very convincing and clear picture of senile pigmentary degeneration is presented.

Dr. K. Rieger, Vienna Austria: In closing, in regard to the way in which detachment of the vitreous comes about, one must distinguish sharply between a spontaneous detachment and one that takes place in a globe which has been opened previously. As to the etiology of spontaneous detachment, it is worth while to refer to the papers presented in January and March 1936, at the meeting of the Ophthalmological Society in Vienna (*Klin. Monatsbl. f. Augenh.* 96: 396 and 695, 1936) and to an exhaustive work on the subject which is to appear, shortly. The detection of a detachment of the vitreous after extraction of cataract has been carried out does not permit one to infer anything about an eventual causal relationship with the operation. The detachment may have been present as a myopic or a senile complication before the surgical procedure, without any possibility of ascertaining its presence. My own observations carried out in the examination of that eye of patients with a cataract which still permitted a view of the fundus, speak uniformly in favor of this view. Furthermore, comparative investigations of eyes after intra-capsular and extra-capsular extraction of cataract, respectively, are hardly possible, as the view of the fundus in the latter category is often insufficient for a thorough investigation of the vitreous.

XI. Measurement of Illumination of Optotype Charts. Dr. T. Graff, Rathenow.

Notation of the illumination of test charts is quite insufficient to indicate their luminosity. In the case of charts reflecting the illumination one should state the luminosity per square centimeters, at right angles to the chart, i. e., the density of illumination, in Stilb units, which is, besides, the natural way and the easiest for measurement in the case of translucent test charts. To determine the density of illumination by means of the formula illumination density in apostilb units $=$ illumination intensity in lux units in which 1 stilb $= 10^4 \pi$ apostilbs is too uncertain, as the quality and quantity of light reflected from the chart would have to be considered more carefully. With an illumination density of around 0.05 stilbs and a contrast of the dark figures against the bright background amounting to 99.75 per cent, the influence of fluctuations in illumination and, depending on these, of changes in the diameter of the pupil, is minimal.

XII. Clinical Experience with the New Zeiss Projection Perimeter (According to Maggiore). Dr. H. Serr, Jena.

The reader has carried out many observations on normal subjects and on patients with defects in the central and peripheral field of vision. These observations were checked by simultaneous study with the automatically registering Loew perimeter, made by Jung in Heidelberg, and/or with the Bjerrum screen. Under the experimental conditions chosen, the following results were noted: 1. The determination of the peripheral limits of the visual field with the projection perimeter is effected with surprising ease, so that in the case of children, nervous patients and so on one can frequently get definite and reliable statements, whereas this is not feasible with the Loew perimeter. ·The patients also tired less easily. The peripheral fields for white showed, with the projection perimeter, perceptibly greater extension in the temporal half and below, in normal subjects, than with the Loew perimeter with bright daylight. The projection perimenter meets all the clinical requirements of color perimetry of the peripheral field, but the usual clinical requirements need overhauling from the scientific standpoint. The projection perimeter enables one to determine the peripheral limits of the field with lowered illumination of the test objects, without circumstantial preparatory arrangements, and this may in many cases give data of clinical importance. The instrument is particularly well suited to detect and map out small scotomas in the central visual field and so can well take the place of the Bjerrum screen.

XIII. Can Central Scotomas be Recognized Objectively? Dr. Heinrich Harms, Berlin.

The investigation started from the consideration that defects in the central visual field, if caused by lesions situated between the retina and the geniculate body, would have to go hand in hand, clinically, with a loss of central pupillomotor sensitivity, or at least its diminution, on account of the intimate connection between the visual pathways and the ascending light reflex path. The experimental principle of alter-

nating and varying illumination used by Hess in studying hemianopic pupillary stasis was adapted to the research in question by the construction of suitable opaque diaphragms or shutters (*Blenden*). These permit a change from central to peripheral illumination of the retina and allow one to compare the pupillary reaction which takes place in each case. The extent of this pupillary reaction can be equalized by making the central stimulation field smaller or by lowering the intensity of its illumination. The quotient of the light volumes used centrally and peripherally characterizes the pupillomotor conditions of the eye under examination. Preliminary studies showed that, with a marked degree of constancy in a given eye, the quotient of subjects with normal vision varies greatly in individual cases, viz., from 0.6 to 0.07. Differences between the two eyes of the same person up to one third of the higher value were found. In testing macular degeneration the large majority of subjects were found to have the difference in quotient which was to be expected from the amount of vision present. In some the quotient was found to be greater in the better-seeing eye. This could be explained by differences in the diffusion of light in the refracting media or possibly by separated lesions of the light path and the light reflex path, respectively.

XIV. THE QUESTION OF ACCOMMODATION. DR. H. ERGGELET, Göttingen.

Although the axial length of the eyeball is of basic significance in the question of the origin of accommodation, frequency curves and graphs of ocular length have been lacking up to the present. The curve constructed by E. Tron is based on values for length which have been calculated from the determining sections of the succeeding optical planes in connection with the previously determined state of accommodation of the eye. The reader, together with Geserick, took direct measurements of the anteroposterior diameter of 122 eyes and plotted a curve of frequency. In contrast to the research material of an ophthalmic clinic, a deleterious selection was avoided by using for measurement only the cadaveric eyes from a pathologic institute. Hence one may expect a statistical result which holds good for the population in general. With a group division of 0.5 mm. the curve came out practically symmetrical, but perhaps with somewhat high peaks. The frequency polygon (*Vieleck*) varied very slightly from the binomial curve route. The variation area extended from 21.7 to 28.75 mm. The average axial length amounts to 24.68 ± 0.089 mm. The spread (σ) = ± 0.988. These values were then compared with the figures represented by the curves of Tron.

DISCUSSIONS OF PAPERS XI TO XIV

DR. H. LAUBER, Warsaw, Poland: In order to obtain a uniform illumination of the perimeter arc and to be able to investigate always under the same conditions, the fields should be taken in the dark room, a broad perimeter arc which is illuminated just enough to preserve the light adaptation of the eye being used. By means of a punctal light projection which can be used for determining the peripheral limits of the field, as well as for campimetry with the Bjerrum screen or with

Lloyd's campimeter, the change in position of the test object takes place noiselessly and cannot be in any way checked up by the subject who is being examined. Unfortunately the apparatus has been almost completely relegated to oblivion [sic].

DR. LÖHLEIN, Berlin: Can this method of detecting the presence of a central scotoma by quantitative comparative determination of pupillary reaction be applied also to intelligent children of from 5 to 8 years of age? If that were the case it would throw light on a whole series of basic problems connected with amblyopia due to squint. For instance, if one found no increase in vision after an early and systematic course of fusion training therapy, one could take it for granted that the amblyopia was the primary factor and the strabismus secondary. This conclusion would be essentially corroborated if it could be shown that the light reaction relations between the macula and the periphery, as well, remained uninfluenced. In the other event, that of secondary amblyopia (ex anopsia), the method might allow one to draw conclusions as to whether the site of exclusion due to nonuse is to be sought in the periphery, where optical visual and pupillomotor paths are still united, or centrally. One would suppose that both localizations are possible. Finally, it is not at all unusual to find in the course of fusion training that central vision shows no improvement as measured by the test chart. In spite of this the child who appeared quite helpless on the first day on which the bandage was applied quickly acquires a self-confident and easy bearing. These are evidently cases in which there was no increase in the function of the maculopapillary bundle, while the peripheral portions of the retina gained materially by practice. If anything of this sort does take place, it might perhaps be demonstrated by a corresponding reversal in the relation of the pupillary reaction produced by stimulation of the macular portion of the retina to that produced by stimulation of the peripheral portion. There seems to be a wide field for the application of this new method.

DR. BEST, Dresden: The Zeiss projection perimeter, which I have been using for two months, is unexcelled for clinical purposes. Without going into details, there is one point on which one cannot agree with Serr. It is true that one can map out the most minute scotomas with the projection perimeter, but in this respect the Bjerrum screen is still superior, if the test object is projected on it. This is partially due to the fact that with the screen one can use distances of from 1 to 5 meters and correspondingly smaller test images.

DR. ENGELKING, Heidelberg: The perimeter used by Dr. Serr is undoubtedly useful for many purposes, but I cannot agree with Dr. Best that the instrument cannot be improved on. For one thing, note that the illumination of the perimeter arc is very uneven, as a result of which the white test objects appear relatively much brighter when centrally located than at the periphery. Even cursory inspection shows that the red and/or green markers appear slightly yellow in the periphery and are, accordingly, not invariable. Furthermore, they have not the same luminosity and, again, differ in luminosity from the blue objects. So in studies of the field with these color objects and this perimeter one is testing not the color sense alone, but at the same time contrasts in luminosity, besides which the luminosity of the color objects is not the

same as that of the perimeter arc. These are all requirements which must be met by an up-to-date perimeter. Finally, the physiologic saturation of the two colors of a pair, e. g., the red-green objects, is not the same, so that the limit for green appears more restricted than that for red, which should not occur if persons have a color sense in which green and red are coupled.

Dr. OHM, Bottrop: It is not generally known that opticokinetic nystagmus can be used very well for the objective detection of central scotomas. If the scotoma is large, the revolving drum is screened off more and more from the periphery by means of four screens to a point at which the nystagmus disappears. The course of severe retrobulbar neuritis can in this way be represented in curves of the opticokinetic nystagmus. As a region of 3 degrees is required for the elicitation of this reaction, one has to use another procedure for small scotomas. If, by means of a mirror, one throws a bright image into the eye in such a way that it appears to be in line with the middle of the rotating drum, the opticokinetic nystagmus disappears immediately the moment the light flashes, if it attracts attention sharply. My smallest marker is 0.25 mm. and corresponds to a visual angle of 1.75 minutes of arc. If that does not suffice, larger markers are used until one reaches the point at which opticokinetic nystagmus is inhibited. In this way one can get an objective conception of the visual acuity even in cases in which other methods fail completely, as in the case of a patient with only one eye and a very small pupil with adhesions. The amblyopia of a squinting eye can also be represented by means of opticokinetic nystagmus. In order to make full use of my method, nystagmography, in addition, is requisite.

Dr. PAUL, Lüneburg: Anatomic determination of the axial length of the eyeball by measurement of the eyes of cadavers is of value. The eyes can, however, be fully used only if the total refraction of the anatomically measured eye, as well as that of the cornea, has been previously determined. For the axial length varies greatly not only with varying refraction of the globe but with variation in the composite structure of the optical system and identical refraction.

Dr. H. SERR, Jena: In my comparative studies of central scotomas I have used, in addition to the projection perimeter, the usual technic of the Bjerrum screen with ordinary, i. e., not projected, test objects. Furthermore, I purposely abstained from using any special artificial lighting arrangements, because, for the time being I wished to test the new apparatus only under such conditions as could be easily provided by the practicing physician. In regard to color perimetry, I have already noted sufficiently in my paper that the projection perimeter in its present form meets only the clinical requirements which have been usual up to the present time and not those which must be considered from the scientific standpoint. Dr. Engelking's valid adverse criticism of the apparatus was to be expected, a priori.

Dr. H. HARMS, Berlin: The stimulus for the development of my method was given by occupation with the scotomas of subjects with strabismus. It was intended primarily to determine in this way the location of the point where the strabismic image was suppressed. For

reasons previously stated, opticokinetic nystagmus is not suited to this purpose. The examination of children by my method depends for its success first on whether they have sufficient attention for reliable fixation and then on the development of a method which will insure reliable fixation by the amblyopic eye during the test. A suitable procedure is being worked out. The evident improvement in visual ability of amblyopic children after exclusion of the better eye under a bandage, without any corresponding increase in visual acuity, may perhaps depend on diminution in size of an existing scotoma.

Dr. H. Erggelet, Göttingen: I feel that the output of our measurements would be enriched if we had definite basic and standard values of accommodation. Hence, as was said, I have desisted from building on unreliable figures. As to the value of the graph presented, in studying the problem of refraction no valid conclusions can be reached without exact data on the axial length of the eyeball. In a roundabout way, far-reaching deductions have been made by replacing unknown values by accepted ones in the reckoning. The results have no solid basis. The curves for the axial length of the eyeball, e. g., that of Tron, may be, but are not necessarily, accurate. Research workers should welcome all the more the curves presented, which have a definite basis. They would be unusually valuable for comparison and check-up and would in a given case support the entire study if both curves agreed.

(To be Continued)

Book Reviews

Internal Diseases of the Eye and Atlas of Ophthalmoscopy. By Manuel Uribe Troncoso, M.D., formerly Professor of Ophthalmology, New York Post-Graduate Medical School and Hospital; Instructor in Ophthalmology, College of Physicians and Surgeons, Columbia University; Assistant Ophthalmologist, Presbyterian Hospital, New York. Price, $15. Pp. 530, with 239 engravings, including 95 figures on 82 full-page color plates. Philadelphia: F. A. Davis Company, 1937.

This stately volume is not only a valuable addition to the series of treatises on the examination of the fundus oculi and the accompanying atlases with which the names of Liebreich, Oeller, Haab, Adam, Oatman and many others are connected. It registers, in this reviewer's opinion, a distinct advance in method of presentation, in scope and inclusiveness and in correlation of the ophthalmoscopic findings with the lesions, the clinical history and the symptomatology. With a very practical, as well as a logical, mind, Professor Troncoso has, by his systematic classifications, put one in a position to make a positive diagnosis or at least, as he states, a diagnosis by exclusion. In this respect the chapter on the normal retinal circulation and its disturbances and their relations to local and systemic disease is worthy of special mention as one of the greatest value not only for the student but for the more advanced and experienced ophthalmologist as well. The optical principles involved in the examination of the transparent media and the intra-ocular tissues are explained clearly, succinctly and without recourse to higher mathematics. The technic in these procedures and in others, such as the determination of differences in level, is illustrated by simple diagrams. The chapter on the ophthalmoscope deals, as is usual, with the history of the instrument, but this subject is brought up to date in a welcome description of the latest types of this invaluable aid to ophthalmic diagnosis. The various electrically illuminated models, the binocular ophthalmoscope and ophthalmoscopy with red-free light are considered. There is a valuable section on the physiology and pathology of the field of vision and the various methods of examination in health and disease, including stereoscopic campimetry, angioscotometry and the technic of perimetry. The use of the termination "osis" for hereditary, degenerative, or/and senile changes in contradistinction to inflammatory processes undoubtedly conduces to the logic of teaching and to simplifying classification. Although, as the author modestly states, this terminology has not yet been generally accepted, some ophthalmologists, notably, Mawas, have adopted and made good use of it. The colored plates are well chosen and of uniform excellence in color, drawing and reproduction. In many cases the views of the fundus are supplemented by pictures of microscopic sections of the normal and/or diseased part concerned. Most of these pictures of histologic sections are printed in a position the reverse of that usually shown in textbooks, e. g., with the excavation of the optic disk pointing toward the bottom of the page, the trunk of the nerve pointing upward, and so on. As the lettering is, as usual, upright, this is confusing until one becomes accustomed to it. A special chapter is devoted to an instructive consideration

of pituitary tumors and their sequelae in the form of pressure atrophy, a good survey of this topic being given. In the section on retinal detachment, the material of which is well up to date, an outline is given of the modern operations. Injuries of the choroid, retina and optic nerve so far as they relate to ophthalmoscopy are discussed and described, and there is some comment on the mechanism, etiology and general pathologic features. In a subsequent edition, which may well be called for, one might suggest additional pictures of the fundus illustrative of later stages of retinal arterial obstruction, especially embolism, and of circinate retinitis, Coat's massive exudation, angioid streaks and the condition of the disk in wood alcohol amblyopia.

PERCY FRIDENBERG. ·

Short-Wave Diathermy. By Tibor De Cholnoky, M.D., Associate in Surgery, New York Post-Graduate Medical School and Hospital. Price, $4. Pp. 310, with illustrations. New York, Columbia University Press, 1937.

The subject is treated under the following headings: A historical outline, the physical aspects of short wave diathermy, experiments in bacteriology and on animals, the technic of short wave diathermy and the clinical application.

De Cholnoky emphasizes that it is wrong to say that short wave diathermy is simple and that it carries no dangers; it is not magic but a new agent derived from a form of electric energy. It is not a cure-all and should not be applied unless its physical action is understood and the pathologic condition of the patient to be treated is clear. Heat is the most important result, and hence this treatment is indicated in inflammatory disorders. It is superior to other forms of heat therapy because of its deep action. The specific action on bacteria has not been proved. While diathermy gives more heat on the surface, short wave diathermy allows the penetration of heat and a uniform action in the deeper structures, provided the application of the electrodes is correct. The proper application of the electrodes is most important to prevent the current from concentrating. The subject of short wave diathermy is not well understood, and the contraindications are not yet clearly defined. The great stumbling block has been the inability to measure the degree of heat obtained, and the result can be estimated only by the patient's subjective sensation to heat.

The author finds that short wave diathermy is to be recommended in the treatment of common colds and in sinus disease. For such conditions a special electrode is best.

This is a clearly written description of short wave diathermy and can be recommended to those who are interested in this form of treatment.

Unfortunately, the diseases of the eye are but briefly mentioned. The author apparently has had little experience with the action of this form of therapy on the eye, though the treatment has been carried on in a number of continental clinics. The great difficulty, again, is the inability to judge the degree of heat developed in the interior of the eye, and the possible deleterious effect on the lens.

The chapters on the clinical application will undoubtedly be enlarged and the indications and contraindications defined when more experience has been obtained.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL
INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS
Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

INTERNATIONAL OPHTHALMOLOGIC CONGRESS
Secretary: Dr. E. Marx, Costzeedijk 316 Rotterdam, Netherlands.
Place: Cairo. Time: Dec. 8-14, 1937.

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA
President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN
BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY
President: Dr. P. E. H. Adams, 6 Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING
President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

GERMAN OPHTHALMOLOGICAL SOCIETY
President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

MIDLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. T. E. Ashdown Carr, 34 Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

OPHTHALMOLOGICAL SOCIETY OF EGYPT
President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3 Midan Soliman Pasha, Cairo.
Time: March 1938.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM
President: Mr. Gordon M. Holmes, 9 Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27 Weymouth St., London, W. 1.

OPHTHALMOLOGY SOCIETY OF BOMBAY
President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Time: July 8-10, 1937.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.

Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.

Place: Lindley'a 4, Warszawa.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31 East Southernhay, Exeter, England.

Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris, 7è.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm, Sweden.

Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.

Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.

Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.

Place: San Francisco. Time: June 13-17, 1938.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.

Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Ophthalmological Society

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.

Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.

Place: San Francisco. Time: June 9-11, 1938.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.

Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

Place: New York. Time: Oct. 6-8, 1937.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.

Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Rd., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. William Donoher, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: San Francisco. Time: June 22-25, 1938.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. A. W. Howe, 740 St. Helens Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501—7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Andre Cortopassi, 703 Second National Bank Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY
President: Dr. J. H. Judd, 107 S. 17th St., Omaha, Neb.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.
Place: New Orleans. Time: Nov. 30-Dec. 1-3, 1937.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY
President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Place: Indiana, Pa. Time: Oct. 21, 1937.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY
President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
President: Dr. Charles T. Flynn, 41 Trumbull St., New Haven.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Place: New London. Time: February 1938.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. John King, Thomasville, Ga.
Secretary-Treasurer: Dr. Mason Baird, 384 Peachtree St., Atlanta, Ga.
Place: Augusta. Time: May 1938.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. E. E. Holland, 51 S. 8th St., Richmond.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: April 6, 1938.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. James A. Downing, 406—6th Ave., Des Moines.
Secretary-Treasurer: Dr. O. L. Thorburn, 213½ Main St., Ames.
Place: Des Moines. Time: Nov. 18, 1937.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: New Orleans. Time: Spring, 1938.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. F. N. Smith, Grand Rapids Clinic, Grand Rapids.
Secretary: Dr. Dewey R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. Oram Kline, 414 Cooper St., Camden.
Secretary: Dr. James S. Shipman, 542 Cooper St., Camden.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Marvin F. Jones, 121 E. 60th St., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: May 10, 1938.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. Rosenberger, 221—5th St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514—6th St., Valley City.
Place: Bismarck. Time: May 1938.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. George Truluck, Orangeburg.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.
Place: Columbia. Time: Nov. 9, 1937.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Kate Savage Zerfoss, 165—8th Ave. N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.
Place: Nashville. Time: April 12-13, 1938.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. M. H. Hood, 505 Washington St., Portsmouth.
Secretary-Treasurer: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. James A. Fisher, 501 Grand Ave., Asbury Park, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, 106 S. Main St., Akron.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Secretary: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third
Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Thomas D. Allen, 122 S. Michigan Blvd., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third
Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15
p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Webb Chamberlin, 7405 Detroit Ave., Cleveland.
Secretary: Dr. Walter H. Rieger, Hanna Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. C. E. Silbernagle, 247 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. M. Johnson, 327 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparal St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Lester H. Quinn, 4105 Live Oak, Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. J. D. Carroll, 102—3rd St., Troy, N. Y.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. L. Allen, 1215 Walker Ave., Houston, Texas
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. C. P. Clark, 24 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. O. S. Gilliland, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. F. C. Hertzog, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Guy Maness, Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Society for Clinical Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Jesse Stark, 45 Park Ave., New York.
Place: Squibb Hall, 745 5th Ave. Time: 7 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. A. J. Young, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Ophthalmological Society of the University of Pittsburgh

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

Passaic-Bergen Ophthalmological Club

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October·to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Raymond A. Lower, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto, Canada.
Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington. D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 Eye St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

CHIASMAL SYNDROMES PRODUCED BY LESIONS IN THE POSTERIOR FOSSA

HENRY P. WAGENER, M.D.

AND

PAUL L. CUSICK, M.D.

ROCHESTER, MINN.

The symptoms of lesions producing pressure on the optic chiasm are extremely variable. While various bitemporal hemianopic defects are the most frequent and most characteristic type of changes found in the perimetric fields, asymmetrical or incongruous altitudinal anopsias and homonymous hemianopias are frequently encountered. Unilateral or bilateral complete amaurosis is ascribed usually to a lesion of the optic nerve proper, and in the absence of an ophthalmoscopically visible explanation for its presence it is usually interpreted as evidence of a lesion in the chiasm or in the optic nerve anterior to the chiasm.

In association with choked disks, defects in the perimetric fields suggesting pressure on the prechiasmal portions of the optic nerves or on the optic chiasm itself are interpreted usually as evidence of basofrontal tumor, suprasellar tumor of the Rathke pouch type or tumor of the third ventricle. Ordinarily these diagnoses will be correct. Occasionally, however, evidence in the perimetric fields of pressure on the chiasm will be produced by tumors or inflammatory lesions at some distance from the optic chiasm. It is important to recognize this possibility, since an error in localizing the tumor may logically occur, particularly in the absence of other symptoms or neurologic findings which definitely indicate the location of the lesion.

In 1931, in a paper published in collaboration with Learmonth, Lillie and Kernohan [1] reported the finding of defects in the perimetric fields typical of prechiasmal and chiasmal lesions in a case of tumor of the vermis of the cerebellum and in a case of tumor of the acoustic nerve. In the first case, that of a boy aged 11 years, vision was reduced to 3/60 in each eye. Ophthalmoscopic examination revealed bilateral choked disk, the elevation measuring 2 D. in each eye. Studies of the

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1. Learmonth, J. R.; Lillie, W. I., and Kernohan, J. W.: Unusual Surgical Lesions Affecting the Optic Nerves and Chiasm, Am. J. Ophth. **14**:738-749 (Aug.) 1931.

visual fields on the perimeter showed large, bilateral cecocentral scotomas with bitemporal hemianopia for colors. At operation an astrocytic glioma of the vermis of the cerebellum was removed. The authors expressed the opinion that the ophthalmologic changes suggested a tumor of the third ventricle, a suprasellar tumor or a basofrontal tumor at the midline. The findings at operation and the postoperative course, however, showed definitely that the lesion was in the posterior fossa; the ventricular system was obstructed at that point. In the second case, that of a man aged 31 years, vision was 6/10 in the right eye and 6/30 in the left. Ophthalmoscopic examination revealed bilateral choked disk, the elevation measuring 5 D. in each eye. The fields of vision showed for the right eye inferior altitudinal anopsia for colors and for the left eye inferior altitudinal anopsia for both form and color. The clinical findings were those of tumor of the left acoustic nerve. At operation the tumor proved to be a perineural fibroblastoma of the eighth cranial nerve, but it was too extensive to be removed completely. At necropsy the tumor was seen to be confined to the posterior fossa. It had produced extreme compression and distortion of the cerebellum, pons and medulla oblongata, and moderate internal hydrocephalus without excessive pouching of the floor of the third ventricle.

Learmonth, Lillie and Kernohan gave no definite explanation of the mode of development of these changes in the fields of vision. They merely pointed out the possibility that an ophthalmologic syndrome suggesting direct pressure on the optic nerves or the chiasm may occur in cases of tumor in the posterior fossa.

In studying the fields of vision of patients with proved lesions in the posterior fossa, we have encountered a considerable number of cases of defects in the visual fields typical of a chiasmal or a prechiasmal lesion. We have found: (1) a central scotoma in each eye in one case; (2) a unilateral central scotoma with temporal hemianopia in the opposite eye in one case; (3) unilateral amaurosis with temporal hemianopia in the opposite eye in one case; (4) complete and congruous homonymous hemianopia in one case; (5) bilateral amaurosis without visible evidence of secondary atrophy of the optic nerve in one case, and (6) incomplete bitemporal hemianopia in one case. Ophthalmoscopic examination revealed chronic choked disks of varying degree in all the cases. Visible secondary atrophy of the optic nerve was present in each eye in two cases, and in one eye in another case. Ventriculography was performed in five of the six cases, and in all five marked dilatation of the third ventricle was noted. For this reason we began to feel that the defects in the fields might be attributed to pressure on the chiasm by the dilated third ventricle. This opinion was supported further by the findings in the following case.

REPORT OF A CASE

A man aged 38 years was admitted to the clinic on March 12, 1934. The history as given on admission was supplemented by additional data obtained subsequently. He was in good health until March 1930, at which time he began to have attacks of headache accompanied by gastric distress but not by vomiting. Several months later he was examined at a large diagnostic clinic, where a diagnosis of ulcer of the stomach was made. At that time bilateral, early choked disk was noted. Roentgenograms of the head showed no abnormality. A diet was prescribed, and for the succeeding ten months the patient was comfortable except for intermittent frontal headache.

In 1931 the patient again began to have gastric distress in spite of the use of alkalis. Headaches became more frequent and were accompanied at this time

Stretching of the optic chiasm and tracts by pressure exerted by the third ventricle.

by projectile vomiting. This continued intermittently for the next two years. In August 1933 he began to have attacks of unconsciousness; these lasted for about two minutes but were not accompanied by convulsions. In the latter part of 1933 the patient spent three months in a large general hospital. The diagnoses of peptic ulcer and inoperable tumor of the brain were made, and palliative treatment was advised. At this time bilateral, chronic choked disk, the elevation measuring 3 D. in each eye, was noted on ophthalmoscopic examination. The patient's ision was good. The headaches and projectile vomiting had continued. Six weeks before the patient came to the clinic his vision began to fail rapidly, and for three week he had been totally blind.

Examination at the clinic revealed marked weakness and cachexia, with moderate ataxia and a tendency to fall to the right. The patient appeared very ill. He was unable to distinguish light. Ophthalmoscopic examination revealed

bilateral, chronic choked disk, the elevation measuring 3 D. in each eye. No evidence of atrophy of the optic nerve was visible.

A diagnosis of unlocalized tumor of the brain was made, and the patient was advised to enter the hospital immediately. He returned to his hotel, however, and spent a restless night during which he vomited several times. Toward morning he became comatose and died before a physician could be summoned.

At necropsy the skull was observed to be very thin, and there was evidence of increased intracranial pressure. The third ventricle was markedly dilated and had a very thin wall which bulged about 2 cm. below its normal site. This downward pouching of the ventricle had markedly stretched and thinned the optic chiasm (as shown in the figure). The cerebellum was herniated into the foramen magnum. The internal hydrocephalus was due apparently to ependymitis, which had closed the foramen of Luschka and that of Magendie. The sella turcica was considerably eroded. No tumor was seen. Examination of the stomach showed no evidence of ulcer.

<div align="center">COMMENT</div>

In this case choked disks were known to have been present for approximately four years. If the loss of vision had been gradual, it could have been explained readily by atrophy of the optic nerve fibers from long-continued edema. However, the loss of vision progressing to complete blindness in three weeks suggested the possibility of direct pressure on the optic nerves or chiasm, particularly in the absence of ophthalmoscopically visible evidences of atrophy of the optic nerve. That such pressure or its equivalent stretching was present was demonstrated definitely at necropsy. It was also demonstrated definitely that this pressure was exerted by the outpouching of the floor of the third ventricle. Anatomically, the anterior extremity of the third ventricle presents a diverticulum known as the recessus opticus, which extends forward above the optic chiasm. Dilatation of this recess can exert pressure on the chiasm similar to that of a suprasellar tumor or a tumor of the third ventricle. It seems logical to assume that dilatation of this recess may occur along with dilatation of the third ventricle proper as a relatively late effect of obstruction to the ventricular system in the posterior fossa.

It is important to recognize the fact that as a result of this mechanism changes in the visual fields typical of a prechiasmal or a chiasmal lesion may occur along with choked disks in the presence of tumors or inflammatory lesions in the posterior fossa. On the basis of the objective ophthalmologic examination alone it is often possible to differentiate this syndrome from that occurring in association with lesions in the anterior part of the brain. A carefully taken history of the onset of the loss of vision will often permit correct differential diagnosis. In the case of lesions located primarily in the basofrontal or the chiasmal region, loss of vision is always an early, and often the first, symptom noted by the patient. This loss of vision usually precedes by a definite interval the development of symptoms of general increased intracranial

pressure. In the case of lesions located primarily in the posterior fossa, on the other hand, subjective and objective changes in vision are late symptoms. Transient periods of blurring of vision may be associated with the development of choked disks, but permanent and progressive loss of vision associated with demonstrable changes in the fields of vision other than enlargement of the blindspots always follows by a definite interval the general symptoms of increased intracranial pressure, such as headache, vomiting and ataxia. In some of the cases of this type other clinical findings will point so definitely to the proper location of the lesion that the findings from examination of the perimetric fields may be disregarded. In other cases, however, localization of the lesion will seem to depend solely on the changes in the fields of vision. In such cases, even if the defects in the fields are typical of a chiasmal or a prechiasmal lesion, if there is a history definitely indicating an antecedent increase in the intracranial pressure it is wise for the ophthalmologist to advise ventriculographic studies rather than direct exploration of the basofrontal or the chiasmal region.

SUMMARY

A certain number of patients with a tumor or inflammatory lesion in the posterior fossa will show, in addition to choked disks, changes in vision and in the fields of vision which simulate those produced by tumors and inflammatory lesions in the basofrontal region or around the chiasm. These changes in vision and the fields of vision are late, not early, symptoms of lesions in the posterior fossa. They are due apparently to pressure on the optic nerves, the chiasm or the optic tracts near the chiasm by the dilated third ventricle. It is usually possible to distinguish these lesions from tumors in the anterior fossa by a careful taking of the history.

MUSCULAR ANOMALIES AND AMETROPIA

A PRACTICAL CONSIDERATION

JAMES WATSON WHITE, M.D.

NEW YORK

It may not be amiss to consider practically some of the questions that arise daily in the practice of the average ophthalmologist. For the younger ophthalmologists especially, who may not have met the unusual problems and who have been taught to follow the rules given in most works on refraction, the following remarks may be of some aid.

Rules are made to be broken, and the general rules are made more keenly interesting by a study of the exceptions to such rules. Not without a routine test of the muscle balance for distance and for near and with the eye in the six cardinal positions and a test of the powers of convergence and accommodation can unusual conditions be consistently found. Later, if and when glasses are prescribed, it is equally important to make tests to find if the glasses are doing what they are supposed to do or if by chance the condition has been made worse by following the prescribed rules. In any branch of medicine, if a certain course of treatment does not improve a condition the physician does not hesitate to change to other forms of treatment and to look for some complications which may alter or change a diagnosis. This is not done so frequently in cases of errors of refraction associated with muscular anomalies; rather, if the rules say to do so and so, this is done, and if improvement is not made it is the fault of the patient and not of the rules or of the physician who conforms to them. To emphasize these statements, a few conditions will be cited that have been seen either in patients dissatisfied with previous treatment or in patients seen in consultation.

When each patient examined for glasses is considered as presenting a potential problem of combined refraction, accommodation and muscle balance, the whole examination assumes a different aspect.

I wish to emphasize here the relief from the tedium of refraction that many ophthalmologists express and the satisfaction in finding the key to the problem, which has been overlooked by others, possibly not because of lack of knowledge but by failure to look in a routine manner for the less usual features.

Glasses may be given for one or more of three purposes: first, to improve vision; second, to relieve symptoms of asthenopia, headache and the allied symptoms, and third, to correct a muscle imbalance. If there is no muscle imbalance and the patient's only symptom is reduced vision, the question of prescribing glasses depends entirely on the patient's choice of glasses and good vision or of no glasses and reduced vision. The latter choice is made most commonly by women and girls who are myopic but who, having no other symptoms, choose, for cosmetic purposes chiefly, to allow the myopia to go uncorrected. A common theory is that myopia will increase more rapidly if uncorrected and that the accommodation will become progressively worse. With myopia up to 4 D, these results are rarely seen, and many myopes who frankly say that the glasses are worn only at the theater or occasionally for particular distant vision do not show more rapid progress of myopia, do not have the supposed loss of accommodation and do not acquire a muscle imbalance because of nonuse of glasses. A myope with vision of 20/200, which was corrected to 20/20, frankly said that he would use glasses only when clear distant vision was necessary. The correction caused no symptoms, but he did not like the sharply defined vision that the correction gave him. Being an artist, he pictured to me, when questioned, the haze of a late October day. If that is his pleasure and such vision is doing him no harm, why should correction of vision to 20/20 be arbitrary because of a theory? If, however, astigmatism is present, producing symptoms of asthenopia, if the accommodation is subnormal or if exophoria is present and is reduced or more easily controlled when the correction is worn, there is a reason for advising a more constant use of the correction, and full correction or even overcorrection may be beneficial. However, if a myope has esophoria which increases on the addition of minus lenses, it is often necessary to give an undercorrecting lens. Or the myopic correction may be used for distance and no glasses or an undercorrection of 1 or 2 D. used for near work. This may be best accomplished in some cases by the use of bifocal glasses, especially for children who are alternately changing vision, as from blackboard work to desk work.

Some form of exercises in divergence is beneficial in many cases of this condition, and such exercises definitely relieve the asthenopia, although the esophoria may remain the same.

Esophoria and esotropia in hyperopic patients constitute the muscle imbalance most frequently recognized. The patient is generally given a cycloplegic before a test has been made of the muscle balance or of the accommodation. These tests should be made before using a cycloplegic. Then a test of the muscle balance should be made with the eyes under the influence of a cycloplegic to determine the effect of the cycloplegic on the squint. If the cycloplegic has little or no effect on

the squint, little benefit may be expected from glasses for the correction of the hyperopia. If, however, the squint is definitely reduced by the cycloplegic, glasses will likewise reduce the deviation. After the correction has been worn and the accommodation has returned to normal, the muscle balance should be reexamined to determine the exact result produced by the glasses. If little or no improvement is made in two or three months by the use of a full correction, little benefit can be expected from the glasses.

If no other cause of the squint than hypermetropia has been detected in the earlier tests, a careful test should be made of the condition of the external and internal rectus muscles. In many cases in which the hypermetropia is less than 3 D. and especially in which the glasses have little, if any, effect, the real cause is due to hypertense internal rectus muscles or to underacting external rectus muscles. I reported before this section earlier the case of a child who had convergence of from 80 to 90 degrees, which was corrected by the recession of one internal rectus muscle. I think that, without exception, the internal rectus muscle was the largest that I have ever seen. The degree of hypermetropia was 1.50 D., and lenses did not affect the deviation in the least. Had the hypermetropia been greater and the muscle not so strong, the two factors might have combined to produce the squint, which would have varied in proportion to the relation between the two. However, many cases of this condition are observed in which glasses have been given to children as young as 1½ to 2 years of age and have been worn constantly for some years, with no appreciable effect on the squint. A safe rule would be that when correction of the hypermetropia fails, other or additional causes should be looked for.

More and more frequently I find either esophoria or esotropia, or exophoria or exotropia, associated with hyperphoria or hypertropia. The ametropia may be slight or considerable, may be myopic, hyperopic or mixed and may have some or no effect on the amount of deviation. These are the cases in which the results from the use of glasses or, in fact, from any form of treatment are the most disappointing, and there is little benefit until the vertical deviation is controlled surgically or otherwise.

Many of these patients have third grade fusion but much prefer to have either convergence or divergence. A girl aged 7 in one year showed a change from esotropia of 15 centrads for near, with no lateral deviation for distance, to exotropia of 20 centrads for distance, with no lateral deviation for near. There was right hyperphoria of 4 centrads due to a condition of a left superior rectus muscle. She had third grade fusion but preferred not to use it. Resection of the left superior rectus muscle was advised.

I have seen patients with the retraction syndrome and strabismus fixus associated with convergent strabismus wearing glasses in a forlorn hope that these may correct the deviation, and this hope was shared by the ophthalmologist.

Patients with exophoria and exotropia associated with hypermetropia vary widely in their response to treatment. If the theory of convergent strabismus that by relaxation of the accommodation with plus glasses the convergence is relaxed and causes the eyes to diverge held true here, one would expect that plus glasses for a patient with exophoria or exotropia would cause the eyes to diverge still more. This seems not to have occurred in the experience of many ophthalmologists, and it is not unusual to see a patient with exophoria or exotropia without glasses or a patient with esophoria or esotropia without glasses wearing the full correction, which made each condition a definite divergent squint. Such results can be definitely determined in the office, and more or less plus correction can be prescribed as indicated.

With this in mind, what should be done in the case of a Negro boy aged 5½ with marked divergent strabismus the amount of which was estimated by Hirshberg as 80 degrees and with vision of 20/70 in each eye as determined by retinoscopic examination with the eyes under the influence of atropine, the correction for the right eye being a + 8.50 D. lens ⊃ + 0.50 D. cyl. and that for the left being a + 9.50 D. lens ⊃ + 0.50 D. cyl?

One has to consider the racial and personal characteristics. The hypermetropia was so great that the patient became discouraged and refused to make any accommodative effort. A reduction of 2.5 D. gave apparent, although not actual, correction of the squint. An intense fighting personality, as often seen in the Irish, would have produced as great a convergent squint with the same amount of hypermetropia. The personal equation should always be carefully considered.

The personal equation is most important in the correction of presbyopia. I believe it was von Graefe who said "The younger the ophthalmologist, the stronger the addition given the presbyopes." Here personal habits, desires and business necessities should be considered carefully before one judges the strength of the addition to be prescribed.

Before a postgraduate group I suggested that a prepresbyopic ophthalmologist should decide on the strength of the addition without being prejudiced by knowing the patient's age. A person who is not presbyopic cannot appreciate the inconvenience experienced by too strong an addition for near. This depends more on personal preference than on actual age or accommodative powers. A patient aged 75 has accepted for years nothing stronger than a + 1.50 D. lens added to his full correction for distance.

Hypoclosis is frequently overlooked unless a routine test of the accommodation is made. Such was the case in a man of 26 who had marked asthenopia in vision for all near work. Vision was normal with the correction of slight hyperopic astigmatism. The muscle balance was normal except for a slightly remote convergence near point. The accommodation with his correction was from 2.5 to 3 D., with a typical fatigue reaction. Addition of a + 1.50 D. lens brought the accommodation for the near point to 4 + D. This was prescribed in temporary grab fronts to be worn for one week. The symptoms were entirely relieved, and bifocal glasses of the same strength were prescribed. This correction has been worn for two years, with no return of symptoms. The hypoclosis was probably due to a streptococcic infection two years before the patient's first visit, as the asthenopia dated from this illness.

SUMMARY

The kind and amount of ametropia should be determined approximately before the giving of a cycloplegic.

Tests of the accommodation and the muscle balance should likewise be made before a cycloplegic is given.

Tests of the muscle balance and frequently the test of accommodation should be made while the eyes are under the influence of a cycloplegic.

Then, with the eyes under the influence of a cycloplegic, the exact amount of ametropia should be determined.

With these data before one, rules are not necessary, but the condition in each case can be determined on its own merits, depending on the results of the various tests.

Such a procedure sounds more time consuming than it really is, but the results of the time so spent are most satisfactory.

LEVER ACTION OPERATION FOR INTRACAPSULAR EXTRACTION OF CATARACT

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Lever action intracapsular extraction of cataract is a new method; it derives its name from the fact that the lens in its capsule is dislocated and delivered by lever action. The scientific principle of lever action has been employed ever since the time of Archimedes, in 300 B. C., to perform work in which great resistance is offered or to raise a heavy weight with the expenditure of the minimum of power or effort.

In this procedure the lens is anchored by the attachment of its zonule. Some sort of power or effort must be applied so that the lens in its capsule may be dislocated by overcoming the resistance of the zonule. The nature of the power or effort applied in the different methods has been different. The power or force has to be applied directly to the lens or its capsule in some methods and from the outside only in others.

Thus, in Smith's method the power is applied as pressure with the lens hook from the outside on the sclera., This external pressure raises the tension of the vitreous, which breaks the resistance of the zonule. Traction with a spoon, lens hook or loop directly on the lens in its capsule is used in Pagenstecher's method. Traction on the coagulated lens by means of the diathermy needle is employed in Lacarrère's method.

Vacuum suction on the lens in its capsule is used in Barraquer's, Hulen's and Moore's method. Vacuum suction combined with pressure with the lens hook is employed in Green's method. Traction with the capsule forceps combined with pressure from the outside is employed in the Knapp-Török-Elschnig, Verhoeff, Stanculeanu and Sinclair methods.

In all these pressure, traction or suction methods and their combinations the force available for overcoming the resistance of the zonule is directly proportionate to the amount of effort or power employed. But at times the zonule is so firmly attached that the power required to dislocate the lens in its capsule is too much for the eye to bear.

On the other hand, with the lever action method the force available for overcoming the resistance of the zonule is greater in proportion to the amount of effort or power employed. Hence lever action intra-

capsular extraction is the safest of all the existing methods for removal of cataract.

Two silver instruments have been devised for the purpose of lever action: (1) the Hata-Donga (hyalonavicular) bisocketed fulcrum and (2) the mango leaf dislocator.

A lever has been described, in statics, as a rigid bar, straight or curved, which is capable of turning freely about a fixed point of support. This point of support is called the fulcrum. The effort or power is a force applied at any point of the lever so as to turn it about the fulcrum and thus to overcome a resistance or to raise a weight attached at any other point of the lever.
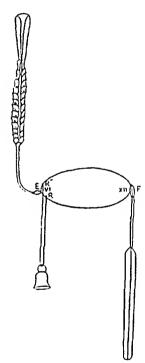
Fig. 1.—Diagram illustrating a lever of class II in the lever action operation for extraction of cataract. F indicates the fulcrum; E, the power or effort, and R and R', the points of resistance or weight.

In the mechanism of the lever action operation for intracapsular extraction of cataract the lens, which has become more or less rigid by the formation of cataract, forms the lever. The resistance of the zonule, attached to the cataractous lens, must be overcome. This is done by fixing the edge of the equator of the lens at the uppermost point, viz., 12 o' clock, by giving the support of the hyalonavicular fulcrum. The effort or power is applied at the diametrically opposite, lowermost point of the equator of the lens, at 6 o'clock, by the mango leaf dislocator so that the cataractous lens in its capsule can turn about the fixed point of support in the hyalonavicular fulcrum.

In a lever of class II, the resistance or weight is placed between the fulcrum and the power or effort.

A lever of class II always acts at a mechanical advantage which is greater than unity.

The hyalonavicular bisocketed fulcrum is a silver instrument which consists essentially of two socket fulcrums: the hyaloid socket fulcrum and the navicular socket fulcrum, carved into the two surfaces of a silver plate. The hyaloid socket is of the shape and size of the hyaloid fossa of the vitreous, measuring about 5 mm. The navicular socket is a boat-shaped socket about 5 mm. long and is made in imitation of a 5 mm. section of the curve of the socket within the ciliary body, into which the equator of the lens in its capsule is socketed by nature. From this socket plate extends a rounded silver rod about 40 mm., long which ends in the handle of the instrument. The handle is a quadrangular silver bar 4 by 2 mm. in cross-section and 80 mm. long.

Nature has been imitated in the design of the instrument in order that lever action can be well enacted. The shape and size of the navicular socket being like the shape and size (5 mm. long) of the socket within the ciliary body, the uppermost margin of the lens in its capsule just fits into the navicular socket fulcrum, and cannot slip when in action, during the first and second stages of the operation. Similarly, the hyaloid socket fulcrum, being like the hyaloid fossa, receives the anterior surface of the lens in its capsule during the third stage of the operation and does not slip.

The mango leaf dislocator is a silver instrument which presents the following parts: a handle, a rod, a bend and a leaf-shaped tip.

The handle is made of silver plate and is 80 mm. long, 10 mm. broad and 2 mm. thick. About three fourths of the two faces is corrugated. A thin straight silver rod 40 mm. long is attached. The rod bends for 10 mm. at a rounded angle of about 90 degrees. The tip of the instrument is leaf-shaped, from which fact it derives its name. The leaf-shaped span is about 4 mm. long and is 3 mm. wide at its widest part. The tip is blunt and about 1 mm. thick. The blunt point cannot penetrate into the bulb of the eye when used on the surface of the eye from the outside.

TECHNIC OF THE OPERATION

After the usual procedures to insure antisepsis and induce anesthesia. a limbal incision into the upper half of the sclerocornea is made. Then the hyalonavicular fulcrum is placed at 12 o'clock on the outer surface of the sclera, and, the lens in its capsule is dislocated by fish-angling jerks applied at 6 o'clock with the mango leaf dislocator from the outside of the sclera. The lens in its capsule is delivered in three stages. The eye is kept bandaged for seven days.

Stage 1 (Dislocation of the Lens in Its Capsule).—The navicular socket is applied at 12 o'clock on the surface of the sclera, outside the insertion of the equator of the lens, and is kept fixed there. The portion of the sclera corresponding to the insertion of the lens in its capsule in the ciliary body can be easily made out, because the position is just behind the periphery of the insertion of the iris, which is visible through the operative cut. The navicular socket should be applied horizontally and tangentially to the optic globe. Thus, the navicular
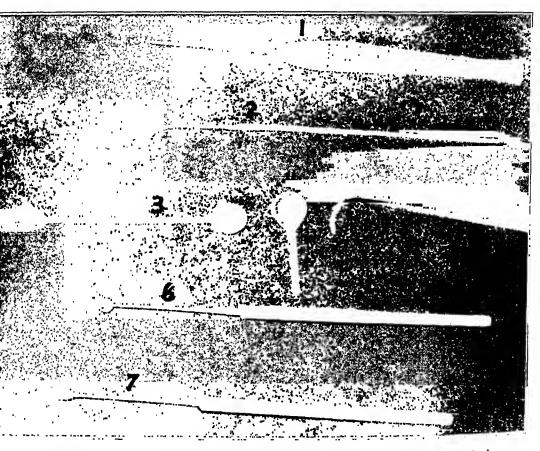
Fig. 2.—*1*, front view of the mango leaf dislocator, one-half the original size; *2*, side view of the mango leaf dislocator, one-half the original size; *3*, navicular face of the hyalonavicular fulcrum, twice the original size; *4*, hyaloid face of the hyalonavicular fulcrum, enlarged ×2; *5*, side view of the mango leaf dislocator, original size; *6*, hyaloid face of the hyalonavicular fulcrum, one-half the original size; *7*, navicular face of the hyalonavicular fulcrum, one-half the original size.

socket forms the fulcrum, into which is socketed the uppermost margin of the equator of the lens in its capsule, padded by the sclera. The lens in its capsule is thus capable of turning in the navicular fulcrum. The navicular socket fulcrum acts automatically, being of the shape and size of nature's own socket.

Now the effort or power is applied as fish-angling jerks by the tip of the mango leaf dislocator at the point diametrically opposite to the point of fixation of the fulcrum. That is to say, the jerky efforts are given at 6 o'clock. These jerks are given from behind forward, on the surface of the sclera, so as to hitch the equator of the lens in its capsule at 6 o'clock. The direction of application of power is therefore away from the vitreous. The jerks are given from 3 mm. behind the limbus at 6 o'clock forward. The tip of the mango leaf dislocator automatically hitches the more or less rigid lower border of the lens in its capsule so as to turn it forward, the navicular socket fulcrum keeping the upper border of the lens in its capsule fixed. These fish-angling efforts act on the resistance of the attachment of the zonule at 6 o'clock by lever action till the resistance is overcome and the zonule is breached at 6 o'clock.

Stage II (Rotation of the Lens in Its Capsule).—After the zonule is breached at 6 o'clock, the lower border of the lens in its capsule is tilted
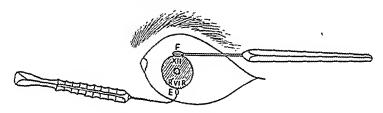
Fig. 3.—Diagram illustrating stage I of the operation, or dislocation of the lens in its capsule. *F* shows the navicular fulcrum, applied at 12 o'clock outside the sclera; *E,* the effort or power applied by the tip of the mango leaf dislocator at 6 o'clock outside the sclera, and *R* and *R'*, the points of resistance of the zonule to be overcome inside on each side of 6 o'clock. The more or less rigid cataractous lens is hereby converted into a lever. In this lever the resistance of the zonule is placed between the fulcrum and the effort; hence a lever of class II is formed (see figure 1).

forward and turns on the navicular fulcrum, which is kept fixed at the upper border of the equator of the lens in its capsule at 12 o'clock, and the second stage of the operation, or rotation of the lens in its capsule, begins. The navicular socket fulcrum is kept fixed on the outer surface of the sclera in the horizontal and tangential position, as before. The tip of the mango leaf dislocator is hitched below and behind the loosened lower margin of the lens in its capsule, the cornea intervening as a pad. By drawing the lower border of the lens in its capsule forward, away from the hyaloid fossa of the vitreous, and tilting it upward with the tip of the mango leaf dislocator, while the upper margin of the equator of the lens in its capsule, padded by the sclera, is fixed within the navicular socket fulcrum, the resistance of the zonule is easily breached by lever action.

The effort required to overcome the resistance of the zonule is less than the strength of the attachment of the zonule. This is because a lever of class II is formed in the mechanism, in which the resistance of the zonule is placed between the fulcrum, at 12 o'clock, and the effort, at 6 o'clock.

In this lever of class II the power or effort, EE', and the weight or resistance at the points R and R' or R" and R''' act on the same side of the fulcrum. F, but the effort acts at a greater distance than the resistance from the fulcrum.
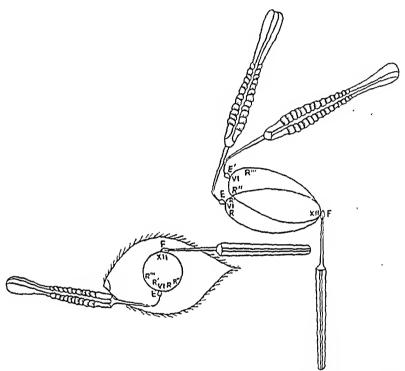
Fig. 4.—Diagrams illustrating stage II of the operation, or rotation of the lens in its capsule. F indicates the navicular fulcrum applied at 12 o'clock, socketing the upper border of the lens in its capsule; E, the effort applied steadily forward by insinuating the tip of the mango leaf dislocator at 6 o'clock on breach of the zonule from the outside to hitch the lower posterior border of the equator of the lens in its capsule so as to tilt it forward, and R and R', the points of resistance in the attachment of the zonule. When R and R' give way, the resistance recedes to the points R" and R'''. The lower border of the lens in its capsule is rotated gradually forward and upward as more and more of the zonule is peeled off.

Mechanical advantage $= \dfrac{FE}{FR \text{ or } FR'}$ or $\dfrac{FE'}{FR'' \text{ or } FR'''} = \dfrac{\text{Arm of effort}}{\text{Arm of resistance}}$.

The arm of effort is the perpendicular distance between the fulcrum and the line of action of the effort or power $=$ FE FE'.

The arm of resistance is the perpendicular distance between the fulcrum and the line of action of weight or resistance $=$ FR, FR', or FR", FR'''.

In a lever of class II, FE is greater than FR and FR', and FE' is greater than FR" and FR"'.

Hence $\frac{FE}{FR}$ or $\frac{FE}{FR'}$ is greater than unity.

Therefore, the mechanical advantage in the lever action operation for intracapsular extraction of cataract is always greater than unity. Again, as more and more of the zonule is breached, the points of resistance at R and R', which at first are at, or close to, 6 o'clock, just beyond which is the point of application of the effort, E, approach nearer the fulcrum, F, at 12 o'clock and move to the point R" and R"'. Thus the arm of resistance FR" and FR"' becomes lesser and lesser than FR and FR'.

Therefore, $\frac{FE'}{FR''}$ or $\frac{FE'}{FR'''}$ is greater than $\frac{FE}{FR}$ or $\frac{FE}{FR'}$.

So the mechanical advantage becomes greater and greater as more and more of the resistance of the zonule is broken. Thus the effort by the mango leaf dislocator required to break the resistance of the zonular attachment is from the first stage of the operation less than, and afterward even still less than, the strength of the attachment of the zonule.
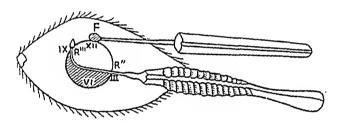
Fig. 5.—Diagram illustrating stage III of the operation. The hyaloid socket fulcrum is applied at 12 o'clock, and the bend of the mango leaf dislocator is applied behind and below the lower border of the lens in its capsule. The remaining portion of the zonule from 6 to 12 o'clock from the points of resistance R'' and R''' up to 12 o'clock is detached gradually as the cataractous lens in its capsule turns on the hyaloid socket fulcrum.

A firmly attached zonule gives way safely and surely on the application of less effort in lever action intracapsular extraction.

As the lower border of the lens in its capsule rotates forward and up to an angle of 45 degrees, i. e., half a right angle on the fixed point of the cataractous lens at 12 o'clock within the navicular socket fulcrum as the result of effort applied at or about 6 o'clock with the mango leaf dislocator, the lower border of the lens in its capsule clears itself of the attached lower half of the cornea, and the third stage, or detachment of the zonule, begins.

Stage III (Detachment of the Zonule).—The lower border of the lens in its capsule has cleared itself of the attached portion of the cornea and has reached the operative cut. The hyaloid socket fulcrum is now placed on the anterior surface of the lens in its capsule. As the lens in its capsule has come out in the open operative wound, the anterior

surface of the lens in its capsule is in direct contact with the hyaloid socket fulcrum. The bend of the mango leaf dislocator is now applied below and beneath the lower border of the lens in its capsule to its posterior side. As the lower border of the lens in its capsule has cleared itself of the attached portion of the cornea, there is no longer an intervening pad of cornea between the lens in its capsule and the mango leaf dislocator. The bend of the mango leaf dislocator is now placed in direct contact with the posterior surface of the lower border of the lens in its capsule. A turning movement is given by the mango leaf dislocator forward and upward to the lower border of the lens in its capsule.

Now that the anterior surface of the lens in its capsule is securely fitted within the hyaloid socket fulcrum and cannot slip and at the same time turning effort is applied to the lower border of the lens in its capsule from the posterior side forward and upward by hitching the bend of the mango leaf dislocator, the lens in its capsule is easily turned forward and upward. Consequently the attached portion of the zonule is detached from below upward completely. The delivery of the lens in its capsule is completed by taking it out at the temporal side of the operative wound.

COMMENT

It may be asked: Why should a new method for the extraction of cataract be launched? What is the necessity of a new method in an era of capsulotomy and of numerous methods of intracapsular extraction? Are there not already so many methods of operation for cataract that the surgeon is sometimes much concerned about which method will be most appropriate in a particular case of cataract?

Those readers who have visited India are aware of the fact that at times the surgeon there has to operate on several hundred persons with cataract in a single day. It is well known that at least 100,000 persons with cataract are operated on in India every year. And still there remain some 500,000 reserves. It will thus be evident that the benefit of extraction of cataract has not yet been universally bestowed, at least in India, and that there are yet hundreds of thousands of human beings living in a state of partial blindness which is curable.

This state of things must be remedied. But how? By popularizing operation for cataract and by enabling the surgeon to operate successfully as soon as there is obstruction of sight.

In order to popularize operation for cataract it must be made safer and surer. Not only this, but it must be made easier, so that a surgeon of ordinary merit can operate well. In order to insure success of the operation at an early stage of formation of cataract, the cataract must be removed intracapsularly. In order to operate by the capsulotomy

method on a cataract, one has to wait till the cataract is mature. Thus the patient has to wait, living in partial blindness, for months or years. Some of the patients may not live long enough to receive the benefit of extraction of cataract. Living in partial blindness, unable to earn a living, bowed down with the weight of cares and anxieties, the patient with cataract goes to an early grave, cursing his ill luck.

This is one of the reasons why I set my heart on finding some solution of the unfortunate problem, a new method of operation by intracapsular extraction which might be universally applicable.

The next question is whether lever action intracapsular extraction answers the purpose for which it is ostentatiously launched. What is there in it to court and claim universality?

In the evolution of surgical intervention for cataract, ever since 1000 B. C., from which date up to the present time there are authenticated records, various methods have evolved. Of these, some have had their day and had to give place to newer and more satisfactory methods. Others are being practiced with more or less success at the present time.

Thus, the depression method of couching anterior·route, inaugurated and practiced by Suśruta,[1] the father of surgical intervention for cataract, with his Yabamukhi Salaka (barley-tipped needle),[2] in which the lens was couched toward the lower inner region of the orbit, had no competitor for a thousand years. In 10 A. D. Celsus, of Rome, introduced his reclination method of couching by the posterior route.[3] But the vitreous was more damaged by the reclination method of couching than by the depression method. Therefore, in 300 A. D., Bagbhat, professor of surgery in Taxilla University, revived the depression method with certain modifications.[4] About 800 A. D. the califs of Bagdad had "Suśruta Sanhita" translated from Sanskrit into Arabic and named it "Ketaba Sushrud." The work both in the original Sanskrit and in its Arabic translation is read as a textbook. Couching was widely practiced till 1748, when Daviel, of France, originated the capsulotomy method.[5] A century later, Alexander Pagenstecher, of Wiesbaden, introduced extraction of the lens in its capsule with a spoon.[6] MacNamara practiced intracapsular operation by pressure of

1. Suśruta Sanhita (dated 1000 B. C.), vol. 7, chap. 17.

2. Mookerji, Girindranath: Surgical Instruments of the Hindus, Calcutta, Calcutta University, 1914, vol. 2.

3. Celsus, cited by Adams, W.: A Practical Inquiry into the Frequent Failure of the Operations of Depression and of the Extraction of Cataract, London, Baldwin, Cradock & Joy, 1817.

4. Bagbhat: Astanga Hriday, Calcutta, B. L. Sen, 300 A. D., vol. 8, chap. 14.

5. Daviel: Sur une méthode de guérir la cataracte par l'extraction du cristalin, Mém. Acad. roy. de chir., Paris 2:337, 1753.

6. Pagenstecher, A.: Ueber Extraction cataractöser Linsen mit ihren Kapsel, Klin. Monatsbl. f. Augenh. 3:316, 1865.

hand and published the pressure method in his book on ophthalmology in 1864. Smith popularized the pressure method which goes by his name and substituted the lens hook for the hand.[7] Barraquer, of Barcelona, introduced the vacuum suction method by his erisphake.[8] But Smith's pressure method and Barraquer's vacuum suction method, though practiced with success by the originator, were not so successful in other surgeons' hands. López Lacarrère has introduced the method of traction on the diathermically coagulated lens in its capsule.[9] Had the pressure method of Smith or the suction method of Barraquer been free from risks and complications, there would have been no need for further developments. But, as is well known, Smith's pressure method is attended with expression of the vitreous and Barraquer's suction method with suction of the vitreous and rupture of the capsule in a large percentage of cases.

Therefore, A. S. and L. D. Green combined Barraquer's suction method with Smith's pressure method in order to limit both suction and pressure. In the Knapp-Török-Elschnig, Verhoeff, Stanculeanu and Sinclair methods, traction with a capsule forceps is combined with Smith's pressure method.

With the induction of akinesia of the facial nerve by Van Lint's method and with the retrobulbar injection of procaine hydrochloride, many operators nowadays are able to operate by the combined methods. The operation is now more successful than before. But the prolonged intra-ocular instrumentation, though not immediately felt by the anesthetized eye, produces postoperative inflammation in many cases. So, although the operation is successful, the end-results are not quite satisfactory. And certainly it is the end-results that count. What is to be done?

All the possible combinations of pressure, suction and traction have been tried but have failed to attain the objective. What is left to do but to adopt some other form of application of force and to apply that force from the outside and in such a fashion that it does not press on or disturb the vitreous and the capsule in any way?

The lever action operation for intracapsular extraction answers the purpose. The effort is applied extra-ocularly and in a direction away from the vitreous. The vitreous is not pressed on or sucked. The capsule is not torn by traction or burst by suction. Hence lever action

7. Smith, H.: Extraction of Cataract in Its Capsule, Arch. Ophth. **33**:64, 1904.

8. Barraquer, J. A.: Quelques indications de la phacoerisis, Clin. opht. **22**:387, 1917-1918.

9. López Lacarrère, J.: Die Electrodiafakie, Klin. Monatsbl. f. Augenh. **88**:778, 1932.

has a claim to universality. And, what is more, by the principle of lever action an extra amount of force is gained to the surgeon's advantage.

Now that I have operated on 300 patients with cataract without rupture of the capsule in a single case and with loss of vitreous in but 1 case, the lever action operation for intracapsular extraction is ready for respectful presentation to ophthalmologists and especially to my young friends. As regards the veterans, no doubt they can operate by any and every method. I told a veteran friend of mine who can boast of having operated on more than 100,000 patients with cataract and has a record of 700 operations in a busy day, "You have magic fingers."

For further particulars about the fish-angling lever action operation for intra-capsular extraction of cataract the reader is referred to the proceedings of the Fifth Annual Conference of the All-India Ophthalmological Society, to be published in September.

EFFECTS OF DINITROPHENOL ON THE PERMEABILITY OF THE CAPSULE OF THE LENS

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AND

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In connection with clinical reports that cataracts occurred in patients who had previously taken dinitrophenol for reducing weight it became of interest to determine whether this drug could alter the permeability of the capsule of the lens in vitro and in vivo. If it were to be assumed that there is some connection between the use of dinitrophenol and the production of cataract it would be necessary to establish some rational mechanism to account for the phenomenon. One possibility, previously suggested by Krause [1] for other types of cataracts, is that dinitrophenol might decrease the permeability of the capsule of the lens and prevent the normal interchange of metabolites. On the other hand, it might increase the permeability so as to allow foreign materials to penetrate into the lens. Such hypotheses have led to fruitful investigations of other types of cataracts, as in the recent study of Gifford and his collaborators,[2] and therefore were of interest in this study. In an attempt to test these possibilities experimentally, the permeability of the capsule of the lens under the influence of dinitrophenol was measured in two ways: (1) in vitro by the utrafiltration method described by Friedenwald [3] and (2) in vivo, with a variety of dyes.

PERMEABILITY OF CAPSULE IN VITRO

Method.—Beef eyes were dissected out at the time of killing of the animals and brought directly to the laboratory. The lens was carefully removed and the capsule separated by making a cruciate incision in the posterior part of the capsule and removing it in a single piece. The capsules were gently shaken with physiologic solution of sodium chloride (0.9 per cent) with repeated changes until all the adherent lens material had been removed. Each capsule was then arranged

1. Krause, A. C.: The Biochemistry of the Eye, Baltimore, Johns Hopkins Press, 1934.

2. Gifford, S. R.; Lebensohn, J. E., and Puntenny, I. S.: The Biochemistry of the Lens: I. The Permeability of the Capsule of the Lens, Arch. Ophth. 8:414 (Sept.) 1932.

3. Friedenwald, J. S.: Permeability of Lens Capsule to Water, Dextrose and Other Sugars, Tr. Am. Ophth. Soc. 28:195, 1930.

to function as an ultrafiltration membrane by being tied over the flared-out end of a piece of 7 mm. glass tubing. Care was used in handling and tying, and a smoothly fitting membrane which did not leak was obtained. To this was attached, by means of a short piece of rubber tubing, a graduated 1 cc. pipet, the whole being held upright by a clamp attached to a stand.

The tube and pipet were filled with a 1:2,500 solution of vital red H.R. in a 0.9 per cent solution of sodium chloride. The membrane on the lower end dipped into a vial containing 20 cc. of the saline solution without the dye. The heights were so adjusted that exactly 10 inches (25.4 cm.) head of pressure was exerted on the membrane. Under these conditions small amounts of fluid and dye were ultrafiltered through the membrane, the rate of filtration varying with the permeability. The volume of fluid passing through could be measured from the fall of the level in the pipet, and the amount of the dye by reading the concentration in the vial colorimetrically against suitable standards. The solution of vital red in some of the tubes contained dinitrophenol in a range of concentrations, the osmotic effect being balanced by adding dinitrophenol in exactly the same concentration to the saline solution in the vial. In this way the only unbalanced forces on each capsule were the osmotic pressure of the vital red and the 10 inch head of pressure of the column of fluid.

In any given experiment eight such ultrafiltration tubes were set up together as a series. These were allowed to stand for twenty-four hours filled with the solution of vital red, to permit the membranes to become saturated with dye and to test for leaks. Then they were carefully refilled with fresh solution of dye, and the amount passing through in the next twenty-four hours was estimated colorimetrically. This value for each lenticular capsule was taken as its control, since the magnitude varied slightly with the inherent permeability of the capsule, its tautness and the area of the membrane exposed for filtration. Then the control solution was carefully removed and replaced by solution of vital red in the same concentration to which dinitrophenol had been added. The first and last tubes were refilled with the solution of vital red alone to serve as controls on the spontaneous change in permeability with lapse of time. The other six tubes were filled with the solution of vital red containing dinitrophenol in concentrations of 1:5,000, 1:25,000, 1:125,000, 1:500,000, 1:2,500,000 and 1:12,500,000, respectively. At the end of twenty-four hours the amount of vital red which had passed through the membranes under the influence of dinitrophenol was read in the colorimeter as before. Interference from the yellow color of the dinitrophenol in the colorimetric readings of the vital red was prevented by adding a few drops of concentrated hydrochloric acid, which changes this indicator to the colorless form. The amount of vital red filtered by each capsule was calculated first as the percentage of the preliminary control value for that capsule, and this value was then calculated as the percentage of the average for the two control capsules which were not exposed to the dinitrophenol. In this way a value was obtained which expressed the change in permeability due to the drug and in which any spontaneous or progressive changes in the capsules had been canceled out. After the period with dinitrophenol, the experiment was run for two additional twenty-four hour periods, the tubes being filled with solution of vital red alone, in order to observe the time required for recovery after the dinitrophenol. As the periods of recovery in all the latter experiments did not differ significantly from the periods for the controls, they need not be considered further.

Results.—The experiments just described were carried through ten times and the average permeabilities in the entire ten series calculated;

the results are shown in the table.   It can be seen that there was apparently a small increase in the permeability of the capsules, the largest change amounting to a 28.2 per cent increase with the 1 : 5,000 concentration of dinitrophenol.   The variability of the results was such that the probable error of the means averaged ± 7.4 per cent, so that there was good probability that the larger changes were not due to chance.

There was, however, one uncontrolled factor in these observations: The solutions of dinitrophenol were made up by successive dilutions of a stock solution, which consisted of a 2 per cent solution of dinitrophenol in a 1 per cent solution of sodium bicarbonate.   The sodium bicarbonate was used to dissolve the dye by forming its soluble sodium salt.   Such a solution has an alkaline reaction and undoubtedly made the capsules in the tubes containing sodium dinitrophenol more alkaline

Amounts of Vital Red Passed by Ultrafiltration Through the Capsule of the Lens with Various Concentrations of Dinitrophenol, Expressed in Average Percentages of the Values for the Controls

		Percentages Filtered with Various Concentrations of Dinitrophenol					
Conditions	Period	1:5,000	1:25,000	1:125,000	1:500,000	1:2,500,000	1:12,500,000
Dinitrophenol in physiologic solution of sodium chloride (average of 10 series)	Dinitrophenol present	128.2	125.6	116.5	113.2	114.0	109.9
	First recovery........	116.5	122.2	111.7	101.9	109.6	101.0
	Second recovery......	106.8	107.0	102.7	97.5	94.5	97.1
Dinitrophenol in phosphate buffer of p_H 7.0 (average of 8 series)	Dinitrophenol present	101.9	99.0	93.7	107.4	93.3	106.2
	First recovery........	98.1	98.4	103.4	100.0	102.8	101.4
	Second recovery......	89.8	95.9	96.6	98.8	105.0	107.3

than those in the tubes containing the control solution.   There was, therefore, the possibility that the increases in permeability were due to a difference in p_H rather than to any specific action of the dinitrophenol. This point was readily checked by running eight more series, in which, instead of the saline solution, an isotonic sodium phosphate buffer of p_H 7.0 was used, thus insuring that both the capsules in the tube containing the control solution and those in the tubes containing solution of dinitrophenol had the same reaction throughout.

In the lower half of the table, where the average results of this second group of experiments are presented, it may be seen that all the results cluster around the 100 per cent value.   All the observed differences were less than the probable errors of the means, so that it is fairly certain there were no changes in permeability produced by the dinitrophenol.   These experiments, therefore, show that if the hydrogen ion concentration is kept constant, dinitrophenol, even in concentrations as

high as 1 : 5,000, does not change the permeability of the capsule of the lens in vitro, which suggests that changes could hardly be expected in vivo.

PERMEABILITY OF CAPSULE IN VIVO

No very satisfactory means of measuring directly the permeability of the capsule of the lens in the living animal are available. Recourse was had to a technic used for demonstrating increased permeability of blood vessels, that is, the injection of dyes. If dinitrophenol increases the permeability of the capsule of the lens, it should facilitate the penetration of dyes into the lens.

Two or three rats at a time were given an injection of 50 mg. of a given dye per kilogram of body weight intraperitoneally. One rat was retained as a control, and the remaining one or two animals were given 20 mg. of dinitrophenol per kilogram subcutaneously. The dose of dinitrophenol was repeated about four hours later, if the rats appeared able to tolerate it. The survivors were killed at the end of six hours, and the lenses of all the rats were removed and carefully examined for the presence of color. A total of forty-nine different dyes, comprising crystalloidal and colloidal and acidic and basic compounds, were thus studied.

Dinitrophenol penetrated into the lens in about one half of the rats given the injections, as could be seen by the faint yellowish color when the lenses were compared with those of the rats not receiving dinitrophenol. In no instance, however, did the test dye penetrate into the lens to a visible degree. There was, therefore, no evidence that dinitrophenol could increase the permeability of the capsule of the lens in vivo sufficiently to let the molecules of dye through. When similar tests are carried out in vascular regions, such as the skin, the dyes pass through the capillary walls and stain the tissues readily in areas of increased permeability.

SUMMARY

Application of dinitrophenol to the capsules of beef lenses in vitro failed to change the permeability of these membranes, as indicated by negative results with the ultrafiltration of a colloidal dye.

Injection of dinitrophenol in rats also did not increase sufficiently the permeability of the lenticular capsules in vivo to allow penetration of the lenses by a wide variety of dyes injected intraperitoneally at the same time.

Accordingly no experimental basis was demonstrated, either in vitro or in vivo, for the assumption that dinitrophenol may cause a change in the permeability of the capsule of the lens of the eye.

SIGNIFICANCE OF THE FOVEAL DEPRESSION

GORDON L. WALLS, Sc.D.

DETROIT

Among vertebrates which are not too strongly nocturnal a local modification of the retina for maximal visual acuity is of widespread occurrence. The modified area (which is single except in some birds) may be small or extensive, a broad horizontal band across the fundus or a suprapapillar crescent, round or in the shape of a variously oriented oval, located centrally or temporally even as far as the periphery and on or slightly above or below the horizontal meridian.

The generic term for such a region of high resolving power is area centralis—misnomer though this name often is. An area centralis may be distinguished from the surrounding retina only by the visual cells being more numerous in it per unit of retinal area. When this massing of visual cells is marked, however, it is usual to find several accompanying adaptations: The visual cells are slenderized, to facilitate their aggregation; they are elongated, to enable them to maintain their volumes and thus their individual limens; they are connected in smaller numbers to bipolar cells, and these in turn are connected in smaller numbers to ganglion cells, so that, the absolute numbers of neurons being increased, local thickening of the nuclear layers occurs; and the ratio of rods to cones is more or less reduced, the rods often being entirely eliminated.

The area centralis at its highest development is thus a complex of adaptations all of which make for increased visual acuity. The cones, which mediate sharper vision because they are less summated in opticus fibers, take precedence over the rods. Their diameters are reduced, and they are tightly packed together. This results in a piling up of the outer nuclear layer, and the other layers are correspondingly thickened and still further augmented by an actual increase in the ratio of ganglion cells to bipolar cells and that of bipolar cells to visual cells.

In a strongly diurnal animal the entire retina may, of course, be organized like an area centralis in some other animal, and one may in this case speak of "universal macularity." On the other hand, the need for a preponderance of rods and a maximal neurologic summation prohibits a strongly nocturnal animal from having an area centralis of any sort, unless the eye is very large.

The area centralis, then, represents a compromise. Animals may produce one if they can afford to devote a portion of the retina to vision of high acuity, yet must retain most of the retina in condition to afford vision of high sensitivity. The adaptations which comprise an area centralis are local phenomena for the simple reason that the same patch of retina cannot be built for the highest visual acuity and for the highest visual sensitivity at the same time.

It might seem that if an animal had evolved a "perfect" area centralis by following the aforementioned rules, there would be nothing that it could do to increase still further, locally, the intrinsic resolving power of its retina. The area may become a pure cone one, and each cone may have a private opticus fiber; but the cones must have *some* diameter and would starve the usual avascular retina if they became too long.

One actually finds that in those animals with the greatest reputations for visual acuity the area centralis is often not a smooth, slightly elevated region but contains a central depression which may be round or oblong and shallow or deep. This depression is the fovea centralis (or fovea temporalis) and is present in some fishes, many reptiles, most birds, and some primates, including man.[1] Where a fovea occurs, another obvious factor increasing visual acuity is sometimes added—intra-ocular color filters of retinal types are especially well developed here,[2] and it has long been believed that the depression itself constitutes an improvement over the simpler, afoveate area centralis.

The foveal depression is created by the centrifugal displacement of the retinal elements in the two neuronic layers, so that the inner nuclear, inner plexiform, and ganglionic layers are virtually absent. The displaced material produces a circumfoveal annular (in the primates, macular) eminence, and the centrifugal course of the elongated foot pieces belonging to the cones of the foveal bouquet creates the "layer of Henle," the fibers of which slowly become shortened and erect to form the more ordinary outer plexiform layer of the extra-areal portion of the retina.

The fovea is thus a "thin spot"; and this common definition calls attention to the attenuation of the retinal layers. It is this attenuation which has been traditionally regarded as the raison d'être of the foveal depression. One commonly reads that removal of tissue from the visual path of the foveal cones permits light to reach them unimpeded. Indeed, the beginner in ophthalmology is likely to assume that the superiority of

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1. See the summary of Kahmann, H.: Ueber das foveale Sehen der Wirbeltiere: I. Ueber die Fovea centralis und die Fovea lateralis bei einigen Wirbeltieren, Arch. f. Ophth. **135**:265, 1936.

2. Walls, G. L., and Judd, H. D.: The Intra-Ocular Colour-Filters of Vertebrates, Brit. J. Ophth. **17**:641 and 705, 1933.

foveal vision is largely due to the excavation of absorptive tissue and to minimize the importance of the other adaptations of the area centralis, or macula.

This accepted theory is predicated on the assumption that the transparency of the retina is appreciably less than that of the vitreous. The perfect clarity of living retinal tissue in situ renders this dubious; but in any case, and particularly in the region which is, to begin with, one of high threshold, little would be gained by taking advantage of the difference in extinction [3] occurring in the thickness of retinal tissue removed as compared with that taking place in the equal thickness of vitreous which naturally fills in the excavation. Again, an afoveate area centralis is always thicker than the unspecialized surrounding retina—in spite of which it is obviously much superior in resolving power, indicating that the production of a thin spot as such is not very important.

Still more serious objection arises from a consideration of the profiles of the highly developed foveae of diurnal sauropsidans. The fovea of a lizard (fig. 1) or that of a bird (fig. 2) is characteristically of what one may call the convexiclivate type, while that of man has an extreme concaviclivate form (fig. 3). The temporal fovea of birds, which is never so well developed as the nasal (central) one, may be continuously concave also, though not as broadly so as that of man. It is easy to see which of these two forms of fovea is the superior—though of course it has not yet been shown here what form has to do with such superiority. Surely a hawk with a million foveal cones per square millimeter would not be expected to have a fovea inferior in all-round efficiency to that of man, and performance indicates quite the opposite. Shallow foveae occur in poor-sighted birds such as the woodpecker, and it is well known that the semidomesticated pigeon has a shallow and variable fovea and the domestic fowl and the owls practically none. In birds, then, shallowness is a mark of foveal degeneracy brought about by the development of nocturnality or of domestication. Similarly, among the reptiles, in which the convexiclivate fovea is customary, there is one form, Sphenodon (fig. 4), in which the secondary adoption of nocturnality [4] has resulted in the foveal depression becoming broadened, shallow and tending toward the concaviclivate type as a step on the path to possible eventual elimination.

In the light of the situations in Sauropsida, the human fovea is thus seen to be actually crude. It either has never been developed to a high

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3. The more detrimental phase of such extinction would of course be scattering, due to optical heterogeneity, rather than absorption; but retinal tissue appears to be quite homogeneous optically.

4. Walls, G. L.: The Reptilian Retina: I. A New Concept of Visual-Cell Evolution, Am. J. Ophth. **17**:892, 1934.
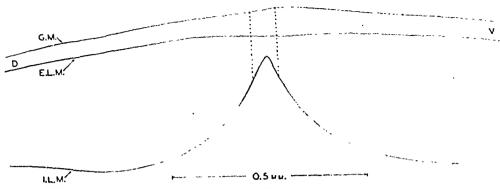
Fig. 1.—Microprojection tracing of the central section through the fovea of a diurnal lizard, Crotaphytus collaris. The portion of the retina between the dotted lines is thinner than the extrafoveal portion of the retina of the fundus generally. *G.M.* indicates the glassy membrane; *E.L.M.*, the external limiting membrane; *I.L.M.*, the internal limiting membrane; *D*, the dorsal side, and *V*, the ventral side.
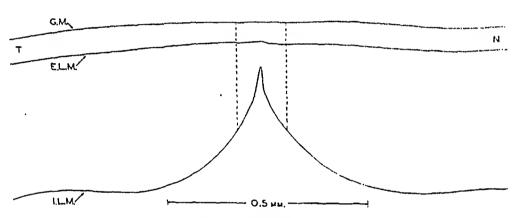
Fig. 2.—Microprojection tracing of the central section through the nasal (central) fovea of a hawk, Buteo b. borealis. The portion of the retina between the dotted lines is thinner than the extrafoveal portion of the retina of the fundus generally. *G.M.* indicates the glassy membrane; *E.L.M.*, the external limiting membrane; *I.L.M.*, the internal limiting membrane; *N*, the nasal side, and *T*, the temporal side.
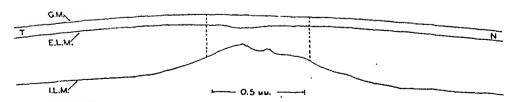
Fig. 3.—Microprojection tracing of the central section through a normal human fovea (from a slide loaned by Dr. Cecil S. O'Brien). The portion of the retina between the dotted lines is thinner than that in the extramacular portion of the fundus generally. *G.M.* indicates the glassy membrane; *E.L.M.*, the external limiting membrane; *I.L.M.*, the internal limiting membrane; *N*, the nasal side, and *T*, the temporal side.

degree or else it is degenerate.[5] Yet if attenuation were desirable per se, one would have to consider the convexiclivate form inferior and should be surprised not to find the foveal depression of the phenomenally sharp-sighted falcons and chameleons shaped somewhat as shown in figure 5. The portions of the foveae shown in figures 1 and 2 which are actually thinner than the respective retinas *outside* of the circumfoveal eminence [6] are proportionately far smaller in area than is the
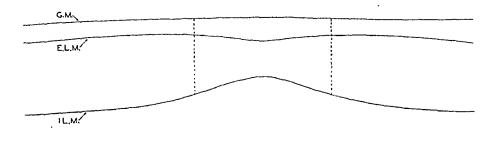
Fig. 4.—Microprojection tracing of the central section through the fovea of a nocturnal reptile, Sphenodon punctatus. The portion of the retina between the dotted lines is thinner than the extrafoveal portion of the retina of the fundus generally. *G.M.* indicates the glassy membrane; *E.L.M.*, the external limiting membrane, and *I.L.M.*, the internal limiting membrane.
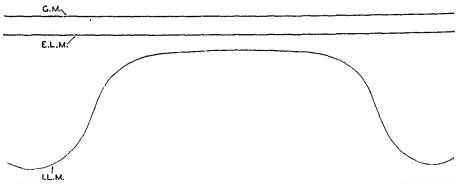
Fig. 5.—General form of foveal profile which would be expected in sharp-sighted birds, lizards and other species if the usual explanation of the foveal depression is assumed to be true.

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5. It would be difficult to determine which, though a knowledge of the situation in the higher apes would help. The irregularities and variability of the human fovea are suspicious, however, for variability often accompanies degeneracy—as in the case of the fovea of the pigeon.

6. The term macular eminence is here avoided, since there is no macula in the strict sense. The much abused term macula lutea refers to coloration, not to conformation. Macular pigmentation is exclusive with the primates, though the avian foveal cones contain only yellow color filters and thus form a yellow spot with a different basis but with a similar significance for vision.

case with the fovea of man and that of Sphenodon (figs. 3 and 4). It is clear that the traditional theory of "removal of tissue" cannot explain the convexiclivate fovea. It is equally clear that one must temporarily dismiss the human fovea and fix attention on the superior sauropsidan type.

If the foveal clivus has some effect other than merely to attenuate the retina locally, that effect must be an optical one. The only optical performance which could affect vision and which can be assigned to the limiting membrane of the clivus is the constitution by that membrane of a refractive surface. The inclusion of the internal limiting membrane among the refractive surfaces of the eye has, naturally enough, never previously been entertained; but there is at least one phenomenon— the familiar foveal reflex of ophthalmoscopy—which might long since have aroused suspicion. The occurrence of this light reflex shows that either the internal limiting membrane or else the entire retina differs in optical density from the vitreous body. But the internal limiting membrane consists of Müller fiber substance, and if the Müller fibers were optically much differentiated from the rest of the retina there would be great detriment to vision. It thus seems logical that the general retinal tissue must differ from the vitreous body in refractive index.

If this difference were such that the retina had the lower index of refraction, total reflection in the deeper part of a convexiclivate fovea would render the central portion of such a fovea blind—which is unthinkable. There are, then, purely logical grounds for believing the retina to have a higher index of refraction than the vitreous body. For the retina generally, this will be of no consequence, because of the virtually perpendicular incidence of the perceived rays; hence there has never seemed to be need for experimental determinations of retinal refractive indexes, and such determinations have never been reported. In a well developed fovea, however, refraction at the clival surface takes on an importance which, in my opinion, is sufficient to account for the evolution of the foveal depression in the areae cantrales.

Figure 6 shows the optical effect of this refraction, a broadening of the retinal image between the two limiting membranes such that a much larger number of visual cells are brought into play than would otherwise be the case. Thus there occurs at the fovea a still further increase in resolving power beyond that produced by the organization of the best possible afoveate area centralis. The increased length of the foveal cones, which was interpreted previously [7] as a compensation for

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7. Walls, G. L.: Human Rods and Cones: The State of Knowledge, Arch. Ophth. **12**:914 (Dec.) 1934. This increase occurs in fishes, reptiles and man but apparently not in birds.

reduced diameter in order to maintain the threshold, may also compensate for the lessened illumination of the expanded image.

If the refraction in the central portion of a fovea were sufficient to increase by 50, 30 or even 20 per cent the number of cones over which the image is spread, it could surely be argued that a very real optical importance had been found for the clival surface and its conformation. In the following analysis, for the sake of this argument an increase of 100 per cent is assumed. If the fovea is assumed to be circular, there need to be determined: (1) to what angle the rays from the lens must be deflected at the clivus in order to double the area of the image at the level of the external limiting membrane [8] and (2) the retinal refractive index demanded by this amount of deflection.

When two circles differ in area by a ratio of 2 : 1, their radii differ by the ratio of $\sqrt{2} : 1$. In figure 6, which shows the same fovea as is
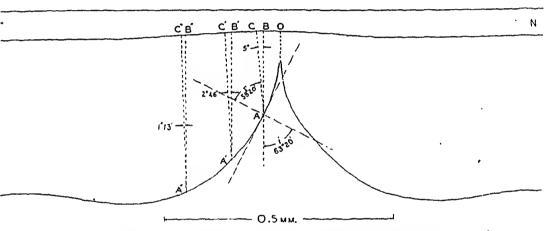
Fig. 6.—Semidiagram showing the enlargement of the retinal image by refraction at the clival surface of the fovea in accordance with the writer's theory. This is the same fovea as in figure 2, reconstructed on the basis of a plane glassy membrane. See the explanation in the text.

shown in figure 2, carefully reconstructed for convenience on the basis of a plane glass membrane, a point, A, has been chosen well down the clivus. A perpendicular from A to the external limiting membrane strikes the latter at B and represents the path that a perceived ray would take through the thickness of the retina if there were no refraction at the clival surface. When OC is laid off equal to $\sqrt{2} \cdot OB$ and AC is drawn, BAC is the angle of deflection required to double the area of an image the diameter of which is $2OB$ at the internal limiting membrane and the center of which is coaxial with O. This angle proves,

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8. There are several reasons for believing that rays entering the bases of the visual cells are confined within the latter by total internal reflection. Hence for the present purpose the external limiting membrane may be taken as the level of perception.

on graphic solution in figure 6, to be 5 degrees. Tangent and normal lines have been drawn at *A*, and the angle of incidence, *i*, is found to measure 63 degrees and 20 minutes. The angle of refraction, *r*, is then 58 degrees and 20 minutes. With the use of 1.336 as the refractive index of the vitreous body in the equation $n_1 \sin i = n_2 \sin r$, the refractive index of the retina, n_2, is found to be 1.403.

Whatever the actual index of the retinal tissue, the increase in the size of the image naturally falls off as the image subtends more and more of the depression. A refractive index of 1.403 being assumed, the angles of deflection at *A'* and *A"* in figure 6 have been measured, the construction lines being omitted for the sake of clarity. *B'A'C'* is an angle of deviation of 2 degrees and 46 minutes, and the ratio *OB' : OC'* is such that there is a 29.80 per cent increase in the area of the image. The corresponding angle *B"A"C"* is 1 degree and 13 minutes, and the area of the image is increased 10.36 per cent. It is not proposed that any valuable deflection occurs at points between *A"* and the crest of the circumfoveal eminence.

It follows that an image confined to the fovea or the foveally perceived portion of any larger image is aberrated by the change in the angle of incidence along the clivus. More prominent aberrations than this are taken care of centrally and eliminated from subjective vision, however. The valuable obtuse angle in the deeper portion of the fovea cannot, of course, be formed abruptly by living tissue.

Though it is unlikely that the human (or any) retina has a refractive index as high as 1.4, and though the human fovea is concaviclivate, there should nevertheless be a valuable expansion of the image even in man. Even the shallow depressions ("incomplete" foveae—Kahmann) seen in some animals should at least have a dispersive effect sufficient to counteract the contractive action on the image of the otherwise convex surface of the area centralis. If one wonders that the majority of foveae, including human foveae, seem poorly formed for the best performance of the function here suggested, it should be borne in mind that most areae centrales are much inferior to those of average birds and lizards in all other respects as well.

It is to be hoped that investigators who have the proper equipment available will soon furnish data as to actual retinal refractive indexes. I am assured that there are applicable methods of determination and that there are no theoretical or practical difficulties which should be insuperable. If retinal tissue proves to have an index to any degree approaching the value of 1.4 stressed for the sake of argument in the foregoing reasoning, it may safely be concluded that the principal function of the foveal depression is the one proposed in this communication. Otherwise, a reasonable explanation of the depression is still to be sought, for the comparative situation shows the traditional theory of the "removal of tissue" to be untenable.

CHANCRE OF THE UPPER EYELID IN AN INFANT TWO MONTHS OF AGE

REPORT OF A CASE

ALFRED APPELBAUM, M.D.

NEW YORK

Although the ocular region is not an uncommon site for extragenital chancres, chancre in an infant and involvement of the upper eyelid are both rare.

Marin [1] reported a case of chancres on the vulva in a girl of 11 months and another case of chancre of the upper lip in a child of 2 years. Foveau de Courmelles [2] reported a case of chancre of the gums in an infant. Bertin, Christin and Lesenne [3] reported a case of chancre of the conjunctival surface of the upper eyelid in a girl of 8 years.

Fox and Machlis [4] reported the case of a colored man who showed a chancre of the upper eyelid eight weeks after being bitten on that structure while fighting. T'Ang and Hu [5] reported a case of conjunctival chancre involving the middle portion of the upper fornix in a Chinese man of 43, who simultaneously presented a penile chancre. Gaté and Genet [6] reported a case of chancre at the internal angle of the eyelids in a man of 26.

Bulkley [7] classified 9,058 cases of chancre reported in the literature and found the eyelids and conjunctiva to be involved in 4 per cent.

1. Marin, A.: Deux cas de syphilis acquise chez le nourrisson, Union méd. du Canada **63**:374-377 (April) 1934.

2. Foveau de Courmelles: Infection syphilitique gingivale par une brosse à dents, J. d. mal. cutan. **17**:705, 1905.

3. Bertin, E.; Christin and Lesenne: Chancre syphilitique de la face conjonctivale de la paupière supérieure, Bull. Soc. franç. de dermat. et syph. **40**:213-215 (Feb.) 1933.

4. Fox, Noah, and Machlis, Samuel: Primary Syphilitic Lesion of the Upper Lid, Am. J. Ophth. **1**:701-702 (Sept.) 1924.

5. T'Ang, T. K., and Hu, C. K.: Chancre of Retrotarsal Fold, Nat. M. J. China **17**:106-109 (Feb.) 1931.

6. Gaté, J., and Genet, L.: Chancre syphilitique de l'angle interne des paupières, Bull. Soc. franç. de dermat. et syph. (Réunion dermat.) **41**:553-555 (April) 1934.

7. Bulkley, L. D.: Syphilis in the Innocent, New York, Bailey & Fairchild, 1894.

Subsequently Muncheimer [8] and Scheuer [9] classified 10,265 and 14,590 cases of chancre, respectively, and verified this percentage.

Maxey [10] studied the reports of a number of cases of primary syphilitic lesions of the eye and noted the frequency of occurrence shown in the tabulation. It appears that the conjunctiva, particularly the bulbar portion, offers Spirochaeta pallida biologic conditions especially conducive to its growth.

The case reported herein is of interest for several reasons: (1) the unusually early age at which infection occurred and the primary lesion appeared, (2) the maternal source of the infection and (3) the negative results of the initial dark field examination of serous material obtained from under the crust of the suspected, untreated lesion.

Site	Number of Cases
1. Conjunctiva	
Upper palpebral	5
Superior fornix	3
Lower palpebral	3
Inferior fornix	3
Location indefinite	13
Plica and tarsus	1
Bulbar	21
2. Lid	
Upper	1
Lower	4
Ciliary border	2
Location indefinite	27
3. Canthus	4
4. Location unknown	2

REPORT OF CASE

History.—W. C., a Puerto Rican boy born Jan. 6, 1937, was brought to the ophthalmic clinic of the Flower-Fifth Avenue Hospital on March 16, 1937. The mother stated that about March 6, two months after birth, she noted a swelling of his right upper lid. After the first few days the swelling began to discharge and then broke into an "open sore." As it increased in size, she became alarmed and sought medical attention. The infant's life was otherwise normal up to that time.
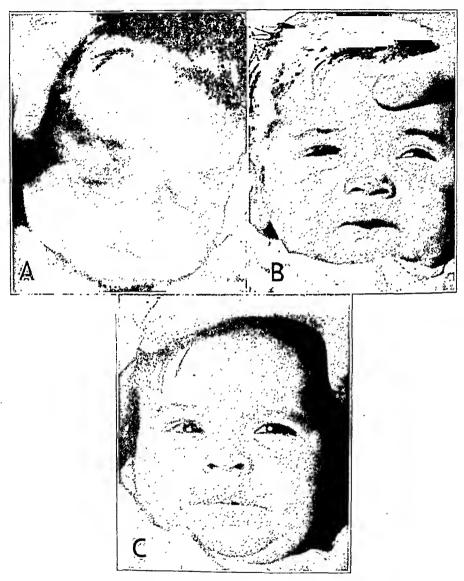
Examination (March 16, 1937).—The outer half of the upper lid of the right eye, the lower lid and the bulbar conjunctiva were perfectly normal. The inner half of the right upper lid, however, presented a sharply defined oval ulcer about 3 mm. wide with its long diameter on the horizontal level, mounted on an induration which appeared to be painless to manipulation. The surface of the ulcer was

8. Muncheimer, F.: Ueber extragenitale Syphilisinfektion, Arch. f. Dermat. u. Syph. **40**:191-235, 1897.

9. Scheuer, O.: Die Syphilis der Unschuldigen, Berlin, Urban & Schwarzenberg, 1910.

10. Maxey, E. E.: Primary Syphilis of Palpebral Conjunctiva, Am. J. Ophth. **1**:13-17 (Jan.) 1918.

uniformly level, dry, crustlike and about 0.5 mm. below the level of the overlying border of the slightly edematous encircling skin. At the free margin of the ulcerous lid was noted some dried-up secretion. The upper palpebral conjunctiva was somewhat injected. The preauricular glands on the right side were enlarged and palpable.

A, appearance of the chancre on the patient's admission to the hospital; *B,* appearance of the chancre one week after local and general treatment was instituted; *C,* appearance of the right lid after healing of the chancre, which left a notched defect in the margin of the lid.

A smear showed no organisms, but many pus cells were observed. Dark field examination of serous material removed from under the crust gave negative results. Specimens for both these tests were taken at the time of the first examination, when I strongly suspected a chancre.

Cleansing solutions were prescribed, and the mother was instructed to return with the infant on the next clinic day, March 19. On her return to the clinic the discharge at the margin of the lid was noted to be more moderate in quantity. Another dark field examination was ordered. This time spirochetes were observed. The child was isolated at the Metropolitan Hospital the next day.

Examination made at the hospital confirmed the diagnosis of chancre. Spirochetes were again demonstrated in the ulcer by dark field examination on March 20. The ulcer became moist and presented a brownish red crusty base. A serosanguineous discharge was present, and many spirochetes were noted in it. The injection of the upper palpebral conjunctiva persisted. General examination of the infant gave negative results excepting for the preauricular glands on the right side, which became markedly enlarged. The results of urinalysis and of a blood count were negative. The Wassermann test of the blood was strongly positive—four plus.

Treatment.—This was carried out in the isolation ward of the Metropolitan Hospital, New York, under the direction of Drs. Van Alstyne Cornell and Girsch Astrachan.

A 10 per cent emulsion of bismuth subsalicylate in oil was administered intramuscularly in three initial injections, in doses of 0.08, 0.1 and 0.13 cc., in the order stated at intervals of three or four days. This treatment was followed by the concurrent administration of mapharsen, injected intramuscularly, and bismuth subsalicylate in oil, injected intramuscularly, each given once a week for fifteen weeks.

The initial dose of mapharsen was 1 mg., and this was increased each week by 0.5 mg. until a dose of 5 mg. was reached; this dose was then maintained. The dose of the bismuth preparation was maintained uniformly at 0.17 cc.

The infant appeared to bear the treatment well and gained the usual weight. No untoward effects were noted at any time, and no complications resulted. While the chancre was completely healed at the close of this course of treatment, the Wassermann reaction of the blood remained unchanged.

The age of the patient, solely, made it unnecessary clinically to differentiate the lesion from a gumma. Yet it must be borne in mind that the latter may commence as an indolent tumor resembling a chalazion or a hordeolum but soon breaks down into an indurated, painless ulcer which tends to invade the deeper structures of the lid, sometimes going on to perforation of the tarsus.

The source of infection in this case is exceedingly interesting. With the cooperation of the Department of Health of the City of New York the following facts were disclosed:

The father of the baby had indulged in extramarital intercourse about a year and a half before, and a penile "sore" developed several weeks later. He received no treatment for five weeks and then placed himself under the care of a physician, on Dec. 10, 1935. The Wassermann reaction of his blood was found to be strongly positive—four plus. After being under treatment for one month, the father brought his wife to the same physician for examination. Her blood was found to show a three plus Wassermann reaction. During the period of Dec. 10, 1935, to Aug. 25, 1936 (eight and one-half months), the

father had received a total of fifteen injections of neoarsphenamine (0.6 Gm. each) and sixteen injections of bismuth subsalicylate in oil, at irregular intervals. The mother had received two injections weekly, bismarsen and bismuth subsalicylate in oil being given alternately, during the period of Jan. 10 to May 11, 1936 (until about the beginning of her pregnancy), and then she became irregular in her attendance. She had had only four injections of bismarsen and five of bismuth subsalicylate in oil during the entire prenatal period. The total amount of treatment received by her during the period of January 10 to August 25 included two injections of neoarsphenamine, fifteen of bismarsen and seventeen of bismuth subsalicylate in oil.

The records of the Flower-Fifth Avenue Hospital revealed the following points in connection with this case : The mother, aged 22, gave a history of one miscarriage of unknown date but no history of syphilis. The Wassermann and Kahn reactions of her blood on Aug. 12 and Sept. 30, 1936 (just before and after the midterm), were negative. On Jan. 6, 1937, she gave birth to a full term normal and healthy-looking child, 7 pounds and 5 ounces (3,317 Gm.) in weight. The placenta was intact, and the Wassermann reaction of blood from the cord was negative. No abnormalities were noted in the mother or· the child on delivery or on discharge from the hospital. A follow-up record, dated three weeks after delivery, likewise showed no abnormalities in the baby, who at that time had gained 1 pound and 7 ounces (652 Gm.) in weight.

On February 2, about one month after delivery, a generalized cutaneous eruption developed in the mother. One month after this eruption she noticed what she described as the "sore" on the right upper eyelid of her baby. Examination on March 24 at the Meinhard Venereal Disease Clinic disclosed that she had a generalized papular cutaneous eruption, which was diagnosed as "late secondary."

<div align="center">COMMENT</div>

Evidently this infant was infected by his mother, who, in turn, had probably contracted the syphilis from her husband. Furthermore, it appears that the infant profited from the limited antisyphilitic treatment received by his mother and was born free from manifestations of hereditary syphilis. Just when and how the infection occurred is a matter of conjecture.

The mother stated with certainty that neither she nor any one else ever kissed the baby; that the infant always enjoyed the use of a freshly laundered towel (not boiled) but she never set aside any towels exclusively for his own use, and that she had no lesions on her breasts and used a nipple shield when nursing the baby.

It is generally accepted that secondary lesions are infectious only when situated on surfaces of mucous membrane where biologic conditions are conducive to the growth of S. pallida, that drying almost immediately destroys the organism, that a break in the skin is not necessary for infection to occur and that the incubation period of a chancre may be as long as ninety days.

Taking into consideration all the facts and findings, I am led to the conclusion that the source of infection in this case was a secondary lesion present in the mother's birth canal at the time of delivery, a prolonged incubation period following the contact.

USE OF IODIZED POPPY-SEED OIL IN OPHTHALMOLOGY

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NEW ORLEANS

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School of Dentistry

Iodized poppy-seed oil, an opaque contrast medium, has been utilized
for the roentgenologic exploration of various cavities and organs of
the body, such as the bronchopulmonary apparatus, the spinal cord, the
paranasal sinuses and the genito-urinary tract. The purpose of the
present investigation was to determine the value of the iodized oil in
the roentgenologic exploration of the tracts and cavities about the eye.
At the same time an attempt was made to ascertain its usefulness as a
therapeutic agent in various diseases of the eye.

Iodized poppy-seed oil is a definite chemical combination of iodine
and poppy-seed oil. It is not a solution of iodine in poppy-seed oil but
represents a stable chemical union between these two substances. The
standard preparation contains 40 per cent pure iodine by weight yet does
not give the ordinary tests for iodine. At room temperature the oil is
viscid, but heating to 35 or 40 C. causes it to become much more fluid,
a property which greatly facilitates injection.

During the past fifteen years a vast literature has accumulated deal-
ing with various uses of iodized poppy-seed oil in diagnostic roentgen-
ology. It is generally admitted that with very few exceptions the oil
may be injected into practically every tissue or cavity of the body,
causing little, if any, pain at the site of injection. In spite of this the
instillation and injection of the iodized oil into the ocular tissues and
spaces were done with the greatest of caution, because of the uncertainty
of the outcome. At first the effects of instillation into the conjunctival
cul-de-sac were observed and recorded. When it was established that
this was a harmless procedure the oil was injected into the periocular
tissues and finally into the vitreous.

INSTILLATION

At the beginning of the experiment the oil was dropped into the
conjunctival cul-de-sac once daily for a period of several weeks. Sub-

sequently this procedure was changed, and an instillation was made four or five times daily for many months, without producing any irritation or annoyance other than slightly blurred vision arising from the oily film on the cornea. The iodized oil did not accumulate in the folds, nor did it remain in the cul-de-sac in sufficient quantity to enable one to obtain a roentgenogram of the surface of the eye. The motion of the lids quickly removed any excess oil from the surface of the eye and thereby made it impossible to procure a roentgenogram. The oil did not irritate the conjunctiva or the cornea; on the contrary, it proved to be a pleasant demulcent.

INJECTION

After it had been determined that no unpleasant effects followed the instillation of iodized poppy-seed oil into the conjunctival cul-de-sac, injection was made into the stroma of the conjunctiva. Although the mesh of the tissue impeded the flow of the iodized oil and broke it into globules, no pain or untoward reaction ensued, in spite of the fact that the oil remained in the tissues for many months.

The next procedure was to inject the iodized oil into the spaces formed by the folds of orbital fascia about the eye. Here, also, the injection was practically painless, and no untoward reaction was produced. In addition, roentgenograms were obtained of the areas into which the injection was made.

Subsequently the oil was injected under the retina and also into the vitreous and anterior chamber. Wherever it was injected, whether into the conjunctiva, into the spaces formed by the orbital fascia, under the retina or into the vitreous, no discomfort followed. In certain tissues and cavities the iodized oil spread; in others it formed globules and gravitated to the lowest point. For instance, when injected under Tenon's capsule it spread out, and when injected into the vitreous it assumed a globular form. The preparation, being an oil, will not mix with the liquids in the vitreous or in the anterior chamber, while the space under the capsule represents a cavity with a pressure which is sometimes less and sometimes greater than atmospheric pressure.

The orbital tissues and spaces into which injection was made are shown in a schematic view (fig. 1). Other figures (figs. 2 and 3) are roentgenograms of the retrobulbar space and the cone of fat.

Into the Episcleral Space.—The injection of this space formed by Tenon's capsule reveals valuable information (figs. 2 and 3). The globe may be effectively visualized in this way, for both lateral and antero-posterior roentgenograms of the eye in its orbit may be obtained. The lateral view appears as if a horizontal section were taken through the eye and orbit. The anteroposterior view gives the appearance of arcs

of densities concentric with the axis of the eye. If an excess of the oil is put under Tenon's capsule, it may be seen coursing in the sheaths of the rectus muscles.

Into the Retro-Ocular Cone of Fat.—After injection of the oil the space of fat which is formed by the rectus muscles and the connective tissue between them resembles an opaque triangular plaque when viewed anteriorly, particularly when the eye is turned up or down, away from the plane. In the lateral view this is, of course, different in shape, conforming to the position of the eye.

Under the Retina.—When injected under the retina the oil does not spread out in a film but assumes a globular shape and travels to the lowest level of the eye. The intra-ocular pressure is not sufficient to
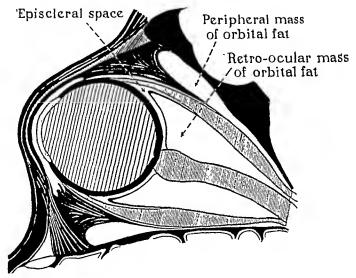
Fig. 1.—Schematic view of the tissues and spaces subjected to injection of the oil.

flatten this globule of oil, and furthermore the oil will not mix with the liquid under the detached retina.

In Cases of Detachment of the Retina.—Iodized poppy-seed oil was injected under the retina in two cases of retinal detachment. In the first case the subretinal fluid was not removed before injection. In the second case the fluid was removed with the hope that the oil would spread out instead of assuming a globular form at the lowest point. In each case, however, it remained in globular form in the eye for many months. Subsequently, multiple punctures were made through the sclera, and the retina became reattached.

Into the Vitreous.—Injections of iodized poppy-seed oil into the vitreous were first performed on rabbits. Only after it was demonstrated that this was innocuous were injections of the oil made into the human vitreous. The oil in the vitreous of the rabbit's eye is shown in figure 5.

After extraction of a cataract large quantities of vitreous were lost. The suture which had been inserted before the extraction was then tied, and the iodized oil was injected to replace the lost vitreous. However, the oil was retained in the eye with great difficulty, and that which remained did not seem to serve any useful purpose.
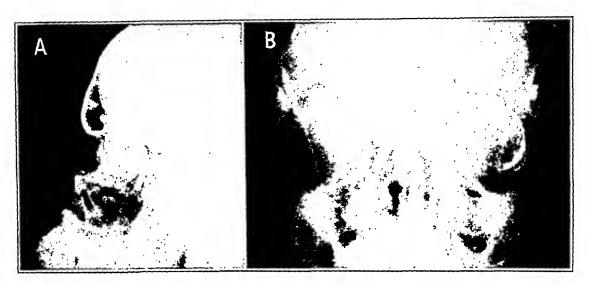
Fig. 2.—*A*, lateral roentgenogram of the cranium, showing to what extent the eye can be outlined in the orbit after the opaque oil has been injected into the episcleral space (Tenon's space). *B*, anteroposterior roentgenogram of the skull, showing arcs of densities concentric with the axis of the eye after the opaque oil was injected into the episcleral space (Tenon's space).

Fig. 3.—*A*, lateral roentgenogram of the skull, showing the retro-ocular space of fat after injection of the opaque oil. The apex is toward the bony optic foramen. *B*, anteroposterior roentgenogram of the retro-ocular space of fat after injection of the opaque oil.

IODIZED POPPY-SEED OIL AS A THERAPEUTIC AGENT

The most obvious benefit from the use of the iodized oil occurred in cases of blepharitis marginalis and in some cases of conjunctivitis.

Fig. 4.—Roentgenogram showing the oil under a detached retina.

Fig. 5.—Roentgenograms taken after the iodized oil was injected into the vitreous.

In the cases of inflammation of the lid the scabs and crusts readily disappeared. In cases of pemphigus of the conjunctiva the oil proved most valuable, and recovery was expedited. Small corneal ulcers responded favorably, whereas larger ulcers showed no improvement.

Because of the large iodine content of iodized poppy-seed oil, patients with incipient cataract were treated with instillations of the oil into the conjunctival culdesac. The treatment was continued for many months
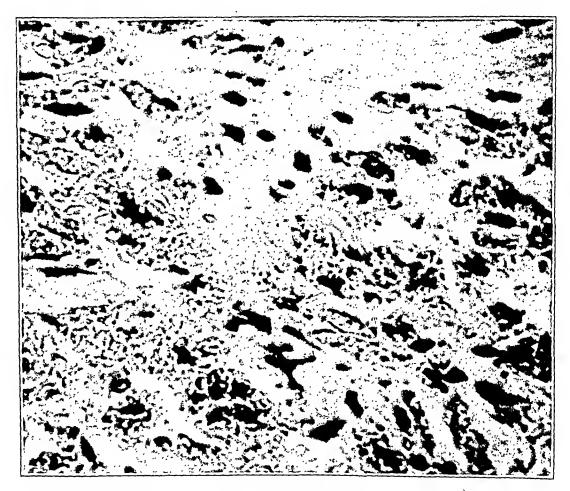
Fig. 6.—Histologic picture of the tissue reaction following the injection of the oil into the pterygium.

with the hope that the iodine would eventually be liberated. It is not quite possible to say that this occurred. However, in one case of detached retina, after a period of several weeks the injected oil changed in color. In another case the oil hastened the ripening of the lens. In a previous paper I [1] have demonstrated that iodized poppy-seed oil is a valuable adjunct in the operative treatment of pterygium.

1. Dimitry, T. J.: Iodized Oil Technic in Pterygium Cases, New York State J. Med. **37**: 719 (April 15) 1937.

HISTOLOGIC OBSERVATIONS

The nature of the tissue reaction following an injection of iodized poppy-seed oil is shown .in figure 6. At the present time reports of histologic examination of only two specimens are available. The first specimen was a section of pterygium into which the oil had been injected and which was subsequently removed. The second was a section taken through the vitreous of a rabbit some time after the injection of the oil.

CONCLUSIONS

Iodized poppy-seed oil is a valuable opaque medium for use in obtaining roentgenograms of the eye and the periocular spaces and tissues.

The iodized oil has a definite therapeutic value in certain diseases of the eye.

No untoward reactions or toxic effects of any kind were observed in the investigation.

Dr. A. Granger, roentgenologist at the Louisiana State Hospital, assisted in this study.

A CASE OF MIXED TUMOR OF THE LACRIMAL GLAND

WITH RETINAL FOLDS AND CHOROIDAL DETACHMENT, WHICH DISAPPEARED AFTER REMOVAL OF THE GROWTH

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So-called mixed tumor of the lacrimal gland is relatively uncommon, and to date about 275 cases of this condition have been recorded. Though the lacrimal gland is almost identical in histologic structure with the parotid and salivary glands, it appears to be much less subject to neoplastic growths than are those glands, which are situated nearer the alimentary tract. The more protected situation of the lacrimal gland as regards trauma or toxic influences suggests itself as a reason for its greater immunity.

Warthin[1] in 1901 made the first complete pathologic investigation of such a tumor. In 1905 Verhoeff[2] reported in detail 1 case of his own and 4 cases collected from the records of the Massachusetts Charitable Eye and Ear Hospital. In 1922 Lane,[3] of the Mayo Clinic, gave a short résumé of tumors of the lacrimal gland reported to that date, about 212 cases in all. Davies[4] in 1934 described 2 cases. Dr. Arnold Knapp removed one such tumor in a case reported by Dr. Last[5] in the ARCHIVES.

Warthin was of the opinion that such tumors are of endothelial origin. However, Verhoeff, Ewing,[6] Samuels and a majority of pathol-

1. Warthin, A. S.: A Case of Endothelioma of the Lacrimal Gland (Myxo-Chondro-Endothelioma Cylindromatodes) with an Analysis of Previously Reported Cases of Lacrymal Gland Tumors, Arch. Ophth. **30**:601, 1901.

2. Verhoeff, F. H.: The Mixed Tumors of the Lacrymal and Salivary Glands, J. M. Research **8**:319, 1904-1905.

3. Lane, L. A.: A Study of Tumors of the Lacrymal Gland with Report of a Mixed Tumor, Am. J. Ophth. **5**:425, 1922.

4. Davies, W. S.: Neoplasms of the Lacrimal Gland, with Report of Two Cases, Arch. Ophth. **12**:33 (July) 1934.

5. Last, Murray A.: Mixed Tumor of the Orbit of the Salivary Gland Type: Successful Removal with Preservation of Eyeball, Arch. Ophth. **13**:812 (May) 1935.

6. Ewing, James: Neoplastic Diseases: A Treatise on Tumors, ed. 2, Philadelphia, W. B. Saunders Company, 1922.

ogists have expressed the belief that they originate from congenital epithelium. The salivary and lacrimal glands are the seat of mixed tumor and are probably derived from the buccal ectoderm, with possible admixture from the maxillary periosteum or the brachial cartilage. They represent elements in the form of strands of cells, alveoli, diffuse masses of mesoblastic tissue, chiefly cartilaginous, mucous and cellular connective tissue. Any of these may predominate, giving rise to nearly pure chondroma, sarcoma or carcinoma, but usually all the types of cells are represented.

There are two points of controversy regarding this type of tumor. The first concerns its origin. It is thought that in the majority of cases this growth arises not from the lacrimal gland but from a fetal rest near the gland. The second question is whether it is truly a mixed tumor. Modern evidence indicates that the areas of mucoid, spindle cell, glandular and other types of tissue may represent a peculiar, unexplained epithelial metaplasia, and hence the term mixed tumor may be a misnomer. It may be more accurate to label the growth in the case reported here "so-called" mixed tumor of the lacrimal gland.

The clinical course of these tumors varies, but in general they are of slow growth, most likely because they usually have a distinct capsule. In the majority of cases this growth has been present long before it is brought to the attention of the ophthalmologist. The size varies, and when the tumor attains some appreciable growth it causes displacement of the eyeball. Lacrimation, as well as edema of the upper lid, is a prominent complaint. Vision decreases with the size of the growth, owing to papillitis, corneal astigmatism or even (rarely) atrophy of the optic nerve.

Most authors assert that mixed tumor of the lacrimal gland is benign. However, Lane reported 12 deaths and 19 recurrences in 95 cases of this condition. Of Verhoeff's 5 patients, 2 required exenteration of the orbit, and 1 died. Of Benedict's 5 patients, who were reported on in 1930, 2 are alive, 2 dead and 1 lost sight of. Mixed tumor shows a low grade of malignancy in the earlier stages and is important then only regionally. But if not completely removed it recurs repeatedly; its growth becomes much more accelerated, and it then definitely shows a much higher grade of malignancy. It is important to save the eyeball only if it is certain that the tumor has been completely removed. Whether a Krönlein operation or a simple incision through the lid is preferable depends on the individual case. However, one gets a bigger field with a Krönlein operation, and as it is important to remove every bit of tumor, that approach is more advisable. Some believe that irradiation should be applied to the site of removal, although Hoche and his

Fig. 1.—Cross-section of the tumor; low power magnification. The growth measured 1¼ by 1 inch (3 by 2.5 cm.) and was enclosed in a capsule 1 mm. thick.

Fig. 2.—Normal lacrimal gland tissue and lacrimal duct structures at the margin of the tumor.
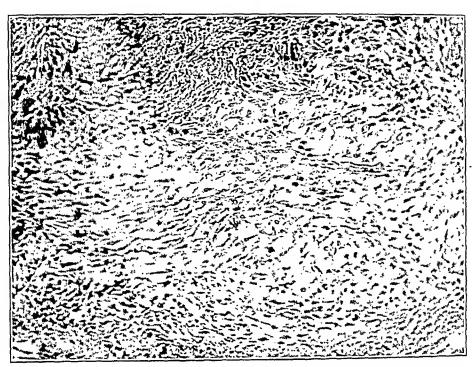
Fig. 3.—Section showing areas of myxomatous tissue and mucoid spindle cell tissue.

Fig. 4.—Section showing areas of pavement epithelium with cystic spaces; occasional mitotic figures; epithelial cells with fine intercellular bridges, and scattered stellate cells.

co-workers [7] stated that this tumor is absolutely refractive to radiotherapy and advise only surgical procedures.

REPORT OF CASE

A married woman 40 years of age was first seen at the Herman Knapp Memorial Eye Hospital one year ago. The complaint was ptosis in the right eye of seven months' duration. There had been tearing and blurred vision of this eye for three months. The past history revealed that twenty years previously the left submaxillary gland had been removed. The cause for this removal was unknown. Otherwise, nothing relevant was learned. Thorough general examination and all the laboratory tests gave negative results. Examination of the right eye showed vision of 20/30— and ptosis. There were limitation of motion of the eyeball up and out and diplopia in the field. The eye became slightly more prominent. There was a mass in the orbit to the inner side of the lacrimal gland, extending nasally and back. It was difficult to determine whether it was attached to the periosteum or not. The fundus presented an interesting picture: The disk was normal. There were seven or eight parallel folds in the retina, extending from the temporal side of the disk to the periphery and concentric with the disk. They resembled the white striae of retinitis often present in a detached retina which has become reattached. Where a retinal vessel passed over the folds there was a slight bend. In addition, in the extreme upper, temporal portion of the periphery there was a choroidal detachment, which was thought by some to be due to a melanosarcoma. Dr. Knapp, in a case which he reported before this section in 1929 under the title "Tumor of the Orbit Producing Retinal Folds," quoted Dimmer's statement to the effect that scleral indentations may raise the choroid in an elevation similar to that of the retina caused by a choroidal growth.

Subsequent events proved that that was the correct explanation, as after I removed the growth by a Krönlein operation in March 1936, both the folds and the detachment cleared up in about eight weeks. This growth was entirely encapsulated. Vision is now 20/20. There is no diplopia or ptosis.

The report of the pathologist, Dr. J. C. Ehrlich, was:

Grossly the tumor measured 1¼ by 1 inch (3 by 2.5 cm.). It was enclosed in a fibrous capsule 1 mm. thick. Histologically there were the following distinct features:

1. Normal lacrimal gland tissue and lacrimal duct structures at the margin of the tumor, outside of the capsule.

2. Areas of myxomatous tissue and mucoid spindle cell tissue.

3. Areas of pavement epithelium with cystic spaces.

4. Occasional mitotic figures in the nucleus of some cells.

5. Epithelial cells with fine intercellular bridges (perhaps the so-called prickle cells).

6. Scattered stellate cells, which may have been precartilaginous cells.

7. Hoche, Baudot, Gault and Legait: Tumeur mixte de la glande lacrymale, Bull. Soc. d'opht. de Paris, April 1935, p. 249.

EUGENIC SIGNIFICANCE OF RETINITIS PIGMENTOSA

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CHARLOTTE, N. C.

Retinitis pigmentosa may be defined briefly as a degeneration of the retina in which "there are atrophy and destruction of the retinal neuro-epithelium, the rods and cones, and their cell bodies"[1] "with hyperplasia of connective tissue and advancement of pigment cells or their granules into the vacant retinal spaces."[2] The natural consequence of this lesion is poor vision, which is aggravated, of course, by poor light.

Retinitis pigmentosa may be inherited as a dominant, a recessive or a sex-linked recessive trait, and this diversity in hereditary pattern is doubtless the reason why the subject is so confused in the medical literature, which shamelessly reveals the unfamiliarity of ophthalmologists with the subject of genetics. Wilmer,[2] for instance, agreed with Nettleship that the ratio of males affected to females affected is 3:2; both dominant and recessive heredity, of course, affect the sexes equally, but in cases of sex-linked recessive traits only the males are affected, and this is doubtless the reason for the preponderance of males with this condition. Wilmer quoted Nettleship to the effect that only thirty-six fathers will transmit the trait to their offspring for every fifty mothers that do likewise. The two sexes transmit dominant traits equally, and parents transmit recessive traits jointly, but in the case of sex-linked recessive traits the affected males transmit only through their daughters, and this is doubtless the explanation for the preponderance of female transmitters. Although different persons even in the same family vary somewhat in the amount of degeneration they inherit to start with, and although nothing is known about the effect of environment on the progress of the lesion, yet the pattern of inheritance is apparently the chief factor in determining the degree of loss of vision and the age when this begins.

1. Friedenwald, J. S., and Chan, E.: Pathogenesis of Retinitis Pigmentosa, Arch. Ophth. **8**:172 (Aug.) 1932.

2. Wilmer, W. H.: Hereditary Factors Responsible for Development of Optic Atrophy and Retinitis Pigmentosa, Arch. Neurol. & Psychiat. **12**:136 (Aug.) 1924.

The first two pedigrees show the inheritance of retinitis pigmentosa as a dominant trait. Those afflicted usually become aware of night blindness between the ages of 25 and 50, sometimes earlier, and the degeneration gradually progresses. But of the thirty-eight persons affected in the two families, only one became too blind to work, and that after 50.

It will be seen in these two pedigrees that retinitis pigmentosa passes down through and affects the sexes equally and that every one affected got it from one parent and passed it on to half the children. Thus it has been inherited as a unitary dominant trait in these two families.

The first three generations of the first family grew up in the era of candle light, and the five women ranging in age from 77 to 97 who gave me the history were very definite about who was night blind or
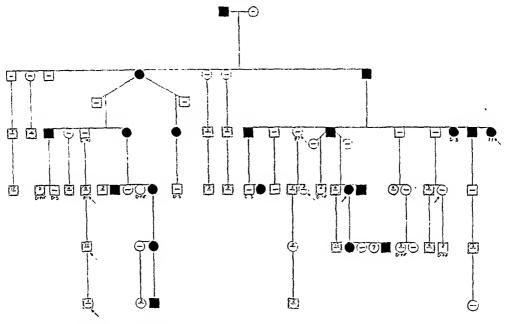
Fig. 1.—Pedigree showing inheritance of retinitis pigmentosa as a dominant trait. In all the pedigrees the squares indicate males and the circles females. *D* indicates died; *D—INF* indicates died in infancy, and *S* indicates single. The squares and circles with a horizontal line show those unaffected, and the black squares and circles show those affected. The numbers below the symbols are the ages in years. The numbers inside the squares, circles and circles within squares indicate the number of children, of both sexes. The arrows indicate members actually seen or examined.

moon eyed in the family and who was not. Reports of ophthalmoscopic examination on the younger generations were secured (members 5, 18, 21 and 26 of the fourth generation, members 3, 6, 7, 8 and 9 of the fifth generation and member 3 of the sixth generation).

The second family had used electric light most of the time, and as many of the members were uncertain whether they were night blind or not it was necessary to carry an ophthalmoscope over two counties and examine them.
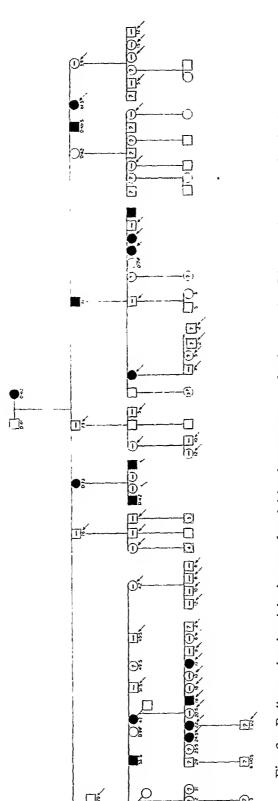
Fig. 2.—Pedigree showing inheritance of retinitis pigmentosa as a dominant trait. The question marks inside the squares or circles indicate that it was not known whether these members were affected.

In the following pedigrees (figs. 3, 4, 5, 6, 7 and 8), which show that retinitis pigmentosa has been inherited as a recessive trait, the affected members became very blind before the age of 25 years. In many pedigrees which show the inheritance of retinitis pigmentosa as a recessive trait the kinship of the parents was close enough and recent enough to be within the knowledge of the descendants, as in the pedigrees shown in figures 3, 4, 5 and 6; in others, kinship of the parents was too remote to be within the knowledge of the descendants, or the marriage brought together members of two unrelated families in both of which retinitis was carried as a recessive trait.

In the family the pedigree of which is shown in figure 3, I examined with the ophthalmoscope the parents, ten of the eleven children and
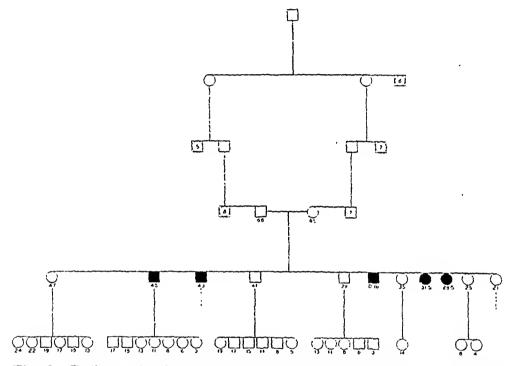
Fig. 3.—Pedigree showing inheritance of retinitis pigmentosa as a receessive trait.

some twenty-five grandchildren. Members over 80 on both sides of the family remembered only one of the kin who had ever gone blind—a man who lost his sight after the age of 75, doubtless from cataract. A woman of 83 years had heard her mother speak of two men, a father and son, who had the same surname as members of the first generation; the father was called "See" Ben and his son "Blind" Ben. "Blind" Ben had been kicked by a horse at the age of 17, but as this was about one hundred and fifty years ago it was not known whether this caused the blindness or exactly what kin these men were to member 1 of the first generation. It seems evident that in this family retinitis pigmentosa was a recessive trait brought into evidence by the marriage of cousins in the third generation.

The first of the two women whose pedigree is shown in figure 4 dated her severe blindness from an attack of malaria at the age of 22; the second sought treatment for poor vision at about the same age. Their parents were first cousins on one side of the family and second cousins on the other.

When the men whose pedigree is shown in figure 5 were choir-boys they had to be led to choir practice on Friday night; their parents were first cousins.

Two of the afflicted members of the family the pedigree of which is shown in figure 6 were seen in a county home, the other two having died
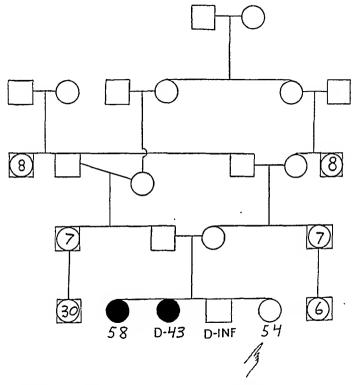
Fig. 4.—Pedigree showing inheritance of retinitis pigmentosa as a recessive trait. The pointing hand indicates a member actually seen or examined.

in county homes. They were too old and garrulous to give any concise statement as to just when they lost useful vision. Their mother was the niece of their father.

The two afflicted members of the family the pedigree of which is shown in figure 7 had been very blind for some years. I examined both parents, who knew little about their grandparents' generation but were not cognizant of any intermarriage or blindness in the family.

The two members with retinitis pigmentosa of the family the pedigree of which is shown in figure 8 became very blind in the second decade of life. I examined the father, who knew of no intermarriage or blindness in the family.

The reason for the difference in the severity of retinitis pigmentosa according to its mode of inheritance seems obvious. When an uncommon condition is inherited as a unit dominant trait, those affected will all be heterozygous for the trait; that is, the trait will be conditioned by a single defective autosomal gene, derived from one parent. But when such a trait is recessive, all those showing it will be homozygous for it; that is, the trait will be conditioned by both members of the pair of defective genes, one derived from each parent.

If this is the proper explanation of the much greater severity and earlier appearance of blindness when retinitis is inherited as a simple
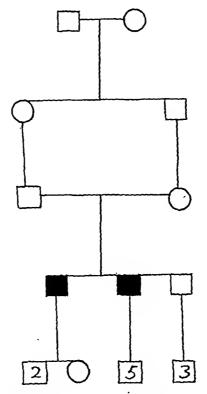
Fig. 5.—Pedigree showing inheritance of retinitis pigmentosa as a recessive trait.

recessive trait, its behavior when inherited as a sex-linked recessive trait is a matter of considerable interest. Will the defective gene on the single x-chromosome of the male behave like the single defective gene, balanced by a normal gene, in dominant heredity, and produce only half blindness or night blindness; will it behave like the pair of defective genes, without any counter-balancing normal genes, in recessive heredity and produce early and severe blindness, or will it behave in an intermediate manner because there is, on the one hand, no normal gene to modify its action and, on the other, no second defective gene to intensify its action? The pedigree shown in figure 9, in which retinitis pigmentosa has been inherited as a sex-linked recessive trait, suggests

that this type of inheritance occupies a place midway between dominant and recessive inheritance. The two affected brothers in the fifth generation, who are still living, noticed impaired vision in the second decade
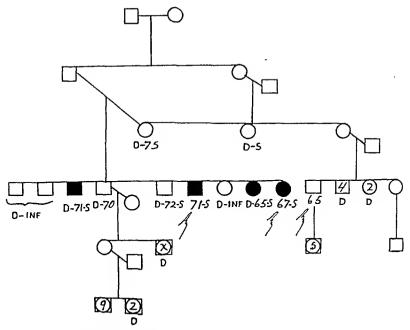
Fig. 6.—Pedigree showing inheritance of retinitis pigmentosa as a recessive trait. The pointing hands indicate members actually seen or examined. The X within the circle in the square indicates that the number of sons was unknown.
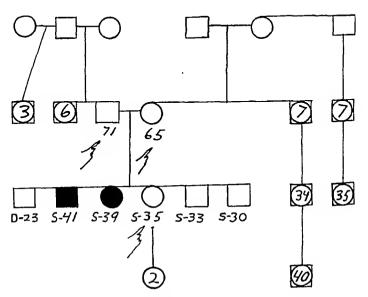
Fig. 7.—Pedigree showing inheritance of retinitis pigmentosa as a recessive trait. The pointing hands indicate members actually seen or examined.

and almost complete blindness by the time they were 45 years old. The brother who died at 21 was noticeably night blind, as is the nephew at 19.

This family illustrates some of the difficulties in dealing with human pedigrees. The mother had only two sibs, a brother who was not blind and a sister who was not a carrier. The mother's mother and all her sibs died early of tuberculosis, so there were no collateral descendants from this generation. The mother's maternal grandmother, who was
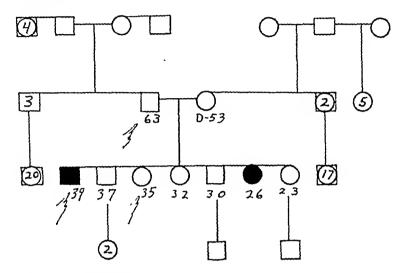
Fig. 8.—Pedigree showing inheritance of retinitis pigmentosa as a recessive trait. The pointing hands indicate members actually seen or examined.
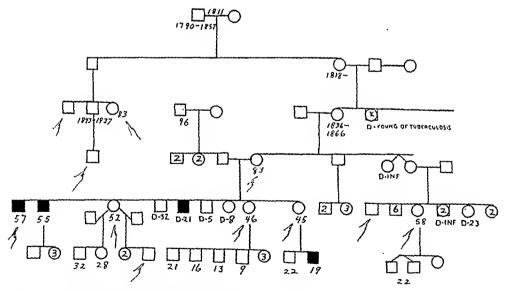
Fig. 9.—Pedigree showing inheritance of retinitis pigmentosa as a sex-linked recessive trait. The pointing hands indicate members actually seen or examined. The X within the circle in the square indicates that the number of sons was unknown.

born in 1818, had sibs, and several descendants of these sibs have been interviewed, but no trace of retinitis pigmentosa was found.

Dr. Wilmer advised that if a child with this lesion is born there should be no more children in that family.

There are various reasons why this advice is not practical. 1. In cases of recessive heredity the number of carriers is twice the number of those who show the trait. 2. In cases of dominant heredity, as regards retinitis pigmentosa, the degeneration may not cause symptoms for twenty or thirty years; member 4 of the third generation of the family the pedigree of which is shown in figure 2, for instance, knew nothing of night blindness in her family until one of her younger girls was big enough to fall over a wheelbarrow one night when going to the barn to milk—and there were already twelve children by that time. 3. Even in the family the pedigree of which is shown in figure 3 it was not until the older children were well along in school that blindness was discovered, and by that time there were already seven or eight children in this family. 4. Ophthalmoscopic examination is not available in the deep country. 5. Examination of children too young to cooperate is very unsatisfactory. 6. In the cases of dominant heredity at least, pigment is probably not present in the early years.

Vision, roughly speaking, depends on two factors—sound eyes and good illumination. Vision will vary directly as both factors are good or bad, but if either factor alone is poor it may be compensated for by excellence in the other factor, and satisfactory vision still result.

In the era of candle light, which lasted until the era of the coal oil lamp, night blindness must have been a severe handicap. In the present era of the electric light, in which electric light is available even in the deep country, the intensity of ordinary household illumination has increased from seventy-five-fold to a hundredfold, and this improvement in light has largely compensated for the defect in eyes heterozygous for retinitis pigmentosa, permitting satisfactory vision in the large majority of cases. Hence good illumination rather than birth control seems necessary when this lesion is inherited as a dominant trait.

When it is inherited as a simple recessive trait, avoidance of marriages of cousins in families which carry the trait becomes imperative, as shown by the pedigrees in figures 3, 4, 5 and 6. The present difficulty with this situation is that there are no family records to show in what families retinitis pigmentosa is carried as a recessive trait. Obviously such records should be started now by the public health authorities in each county, and educational work should be done to prevent hereditary blindness; laws are not necessary to keep women from having blind babies. In the pedigree shown in figure 3, for instance, each of the fifteen sibs of the parents of the blind children has or had one chance in two of carrying the trait, and each of the eighty-four children of these fifteen sibs—the first cousins of the blind children—has had one chance in four of carrying the trait. Each of the six sibs of the blind children has one chance in two of carrying the trait, and each of their twenty children has one chance in

four. The seven children of the blind man will, of course, all be carriers of retinitis pigmentosa, and their children, if any, will have one chance in two of being carriers. Member 1 of the sixth generation in this pedigree married her half first cousin once removed, and another member of this clan married the daughter of member 4 of the fourth generation in pedigree 6, who lived in a nearby district; by the law of chance none of the seventeen children resulting from these two unions are blind, but only ignorance permits the taking of such chances.

When retinitis pigmentosa is inherited as a sex-linked recessive trait, half the sisters and all the daughters of any afflicted man will pass the trait to half their sons. In this situation the responsibility rests largely on the women in the family, and I find from experience in applying negative eugenics that a warning to the women in these families that the chances are 100, 50 or 25 per cent that some of their boys will be blind stops reproduction. Those who are kin to the blind know the weight of this cross.

From the foregoing remarks it is evident that ignorance of the laws of genetics is the prime factor in the continued production of the severer types of retinitis pigmentosa—those seen in instances in which the condition is inherited as a recessive trait or as a sex-linked recessive trait. Plainly, it is the duty of the medical profession to advise members of families with such heredity.

Shall physicians embrace this opportunity to practice preventive medicine by making knowledge of genetics as necessary a part of medical training as knowledge of bacteriology, or shall the blind be allowed to continue to lead the blind?

IMPORTANCE OF DIET IN THE ETIOLOGY AND TREATMENT OF TOBACCO-ALCOHOL AMBLYOPIA

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NEW YORK

It is my purpose to present the results of a clinical investigation which I think is unusual and, judged by older ideas, quite unorthodox. For more than two years patients with tobacco-alcohol amblyopia have been allowed to continue their usual intake of tobacco and alcohol provided they consumed the diet prescribed for them. This work has been carried out slowly and cautiously. All the patients have been hospitalized throughout the period that they were permitted to smoke and drink. The course has been carefully followed by frequent examinations of the visual fields and visual acuity. The daily intake not only of tobacco and alcohol but of every component of their diet over this entire period of hospitalization is accurately known.

First, the previous studies, not made by ophthalmologists, which have a bearing on this subject will be considered. Nine years ago the suggestion was made [1] that vitamin deficiency may be an important factor in the etiology of alcoholic polyneuritis. The following year this suggestion was repeated,[2] and then came reports of cases of alcoholic polyneuritis in which the patient was greatly benefited by the proper diet.[3] In 1931 an internist [4] mentioned that a number of cases of retrobulbar neuritis due to vitamin B deficiency had occurred in Japan, and he expressed the opinion that retrobulbar neuritis occurring in patients with chronic alcoholism should be investigated from this standpoint. In 1933 Spies and De Wolf [5] allowed ten hospitalized patients with the alcoholic type of pellagra to drink between 1 pint (475 cc.) and 1 quart

1. Shattuck, G. C.: Am. J. Trop. Med. 8:539, 1928.

2. Minot, G. R.: Ann. Int. Med. 3:216, 1929.

3. Wechsler, I. S.: M. J. & Rec. 131:441, 1930; Etiology of Polyneuritis, Arch. Neurol. & Psychiat. 29:813 (April) 1933.

4. Keefer, C. S.: . New England J. Med. 205:1086, 1931.

5. Spies, T. D., and De Wolf, H. F.: Am. J. M. Sc. 186:512, 1933.

(945 cc.) of whisky daily, provided they took a well balanced diet plus 75 Gm. of yeast daily. The patients recovered from the pellagra well, despite the large intake of alcohol.

Early in 1935, after a previous study of fifty-five cases [6] of tobacco-alcohol amblyopia and after consideration of the known effects or, better, the lack of effect of alcohol on the nervous system, the present investigation was begun. While the first patient was under treatment a report [7] was published from the Boston City Hospital stating that ten patients with alcoholic polyneuritis had been hospitalized, allowed to consume their usual intake of alcohol, which was between 1 pint and 1 quart of whisky daily, and fed a well balanced, high vitamin diet supplemented with yeast and other vitamin B-containing substances. This was a procedure similar to what I had started with the patients having tobacco-alcohol amblyopia, and it was encouraging to learn that the polyneuritis had improved in every instance and that the rapidity and degree of recovery were apparently just the same in patients who were allowed to drink this large quantity of alcohol as in patients who were forced to abstain from alcohol. In 1934 an investigator [8] in physiologic chemistry calculated what seemed to be the vitamin B requirement of man. Using these calculations, physicians at the Bellevue Hospital [9] in New York have found that every alcohol addict with polyneuritis has had an inadequate intake of vitamin B. There has accumulated a considerable number of reports all tending to indicate the same thing, i. e., that so-called alcoholic polyneuritis is a manifestation of a deficiency disease and is not due to a direct neurotoxic effect of the alcohol.

The present investigation will now be considered. Patients with tobacco-alcohol amblyopia were informed of the nature of their disease and told of the usual treatment for this type of amblyopia, but they were also told that on certain conditions they could enter the hospital without charge to them. The conditions were: (1) that they should continue to smoke and drink as much as they had while contracting the disease, and (2) that they must eat what was given them.

Eight patients have been studied in this way. I shall report on each of them separately. At this time I should like to present the report of one case (case 8) which illustrates the method used and the therapy employed.

6. Carroll, F. D.: Analysis of Fifty-Five Cases of Tobacco-Alcohol Amblyopia, Arch. Ophth. **14**:421 (Sept.) 1935.

7. Straus, M. B.: Am. J. M. Sc. **189**:378, 1935.

8. Cowgill, G. R.: The Vitamin B Requirement of Man, New Haven, Conn., Yale University Press, 1934.

9. Jolliffe, N.; Colbert, C. N., and Joffe, P. M.: Am. J. M. Sc. **191**:515, 1936.

REPORT OF A CASE

CASE 8.—W. H., a 39 year old Negro, was seen by me on Nov. 8, 1935, at the Vanderbilt Clinic. He stated that his vision had been gradually decreasing for seven weeks. He drank between 1 pint and 1 quart of cheap corn whisky daily and smoked one and a half packages of cigarets daily. Vision was 20/200 in each eye, and the fields showed centrocecal scotomas, which are always present in the eyes of persons with tobacco-alcohol amblyopia; the rest of the ocular examination gave negative results. One week later, vision had decreased to 15/200 in the right eye and to 10/200 in the left eye (fig. 1 *A*), and the patient was admitted to the Institute of Ophthalmology. Gastric analysis revealed absence of free hydrochloric acid. Throughout the period of hospitalization he smoked his usual number of cigarets daily and was given between 1 pint and 1 quart of liquor daily. I purchased a quantity of the corn whisky he had been drinking, had it examined chemically and then gave it to him for consumption in the hospital. Chemical analysis failed to show any appreciable quantity of any toxic substance except ordinary ethyl alcohol, and this has been the chemical finding in numerous other samples of liquor used by other patients with this type of amblyopia. The diet which he was given was a high vitamin B, well balanced diet adequate in all respects and supplemented by powdered brewers' yeast [9a] in doses of 2 tablespoonfuls five times daily; vegex (a brewers' yeast extract) in doses of 1 teaspoonful three times daily and wheat germ (Embo) in doses of 4 tablespoonfuls three times daily. He was also given liver extract [9b] intramuscularly in doses of 5 cc. several times weekly and cod liver oil in doses of 1 ounce (30 cc.) daily. This therapy will be referred to later merely as the "prescribed diet."

While the patient was on this prescribed diet and while he was having his usual intake of tobacco and alcohol his vision gradually improved in seven weeks from 15/200 in the right eye and 10/200 in the left eye to 20/25 and 20/30 +, respectively. The fields of vision showed a corresponding improvement, and he was then discharged from the hospital. He was advised to decrease his consumption of alcohol because of any possible effect that such large quantities of alcohol might have on other organs of the body, but he has not followed this advice very well. He was also advised to take 2 tablespoonfuls of powdered brewers' yeast daily, and he has followed this advice somewhat better. Seventeen months have passed since his discharge from the hospital. Vision is 20/20 in each eye (fig. 1 *B*).

COMMENT

All these patients have been followed at least one year. One of the eight patients did not improve. She had atrophy of the papillomacular bundle of the optic nerve, and vision remained stationary. All the other patients showed very satisfactory results, and all have vision for reading. The speed of recovery seemed to be at least as good in these patients as in patients previously studied who abstained from tobacco and alcohol. But I cannot say that this will prove to be true in a larger series of patients. The course of this disease is variable. It has been known that patients may sometimes improve considerably who have merely decreased somewhat their intake of alcohol or tobacco, and infrequently a patient who continued to smoke and drink as much as before

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9a. Budweiser yeast was used.

9b. Solution liver extract-Lilly N. N. R. was employed.

the development of the amblyopia has been known to experience a return to normal vision. But this is most unusual. Certainly, on the basis of previous knowledge of this condition such marked improvement in seven of eight patients who failed to decrease their intake of alcohol or tobacco would never be expected.
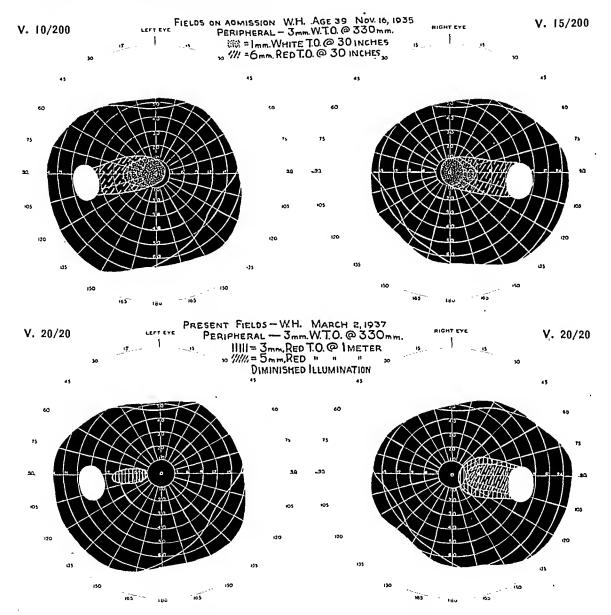
Fig. 1 (case 8).—Fields of W. H.

That the general nutrition of the patient plays a role in the development of this disease is not a new idea.[10] Further, this type of amblyopia

10. Mackenzie, William: A Practical Treatise on the Diseases of the Eye, Philadelphia, Blanchard & Lea, 1855, p. 987. Traquair, H. M.: Edinburgh M. J. **42**:153 (Aug.) 1935. Holth, S.: Acta ophth. **5**:195, 1927. Letchworth, T. W.: Clin. J. **61**:137, 1932. Yudkin, A. M.: Ocular Disturbances Produced in Experimental Animals by Dietary Changes: Clinical Implications, J. A. M. A. **101**:921 (Sept. 16) 1933; Vitamins: Clinical Evidence of Necessity of Vitamins for Function of Ocular Tissue, Arch. Ophth. **14**:112 (July) 1935. Carroll.[6]

is found [11] in patients with alcoholic polyneuritis and in those with the alcoholic type of pellagra, which are almost surely deficiency diseases. In addition, a type of amblyopia occurs in persons with beriberi [12] which is said to be indistinguishable by ocular findings from tobacco-alcohol amblyopia.[13]

Is it justifiable, then, on the basis of all the facts now known, to state at this time that the condition is purely the result of a deficiency state? I do not think so. If that were true, one might reasonably expect to see some other manifestation of deficiency disease in these patients. By the present tests one does not find that in most of these patients. And if that were true, one might also reasonably expect to find a type of amblyopia indistinguishable from tobacco-alcohol amblyopia among patients who do not smoke or drink but who have deficiency conditions such as pellagra. And as has just been stated, there is a possibility that some patients with beriberi do have amblyopia of this type, but at the present time I do not think that there is adequate evidence on this point. Through the agency of numerous physicians in the southern states I recently had the opportunity of examining the eyes of one hundred and ten patients with pellagra who did not smoke or drink in appreciable amounts. None of these patients with pellagra showed any evidence of toxic amblyopia, but numerous patients with pellagra in New York have alcohol amblyopia, and they are all heavy drinkers.

What, then, is the explanation of the production of tobacco-alcohol amblyopia? Perhaps no one can yet give the full answer to that question. Experimentation [14] has shown that animals in a poor state of nutrition are much more susceptible to the injurious action of poisons and toxins than other animals, and it is not unlikely that in the production of this type of amblyopia patients suffering from subclinical nutritional deficiency have a markedly increased susceptibility to tobacco and alcohol. But this is pure theory, and I shall change my theory whenever new facts indicate such change. It is no theory that a series of these

11. Carroll, F. D.: "Alcohol" Amblyopia, Pellagra, Polyneuritis; Report of Ten Cases, Arch. Ophth. **16**:919 (Dec.) 1936.

12. Elliot, R. H.: Tropical Ophthalmology, London, Oxford University Press, 1920, p. 407.

13. Komoto, Jufiro (Tokio, Japan): Personal communication to the author.

14. Rose, S. B.; Rose, W. B., and Kolmer, J. A.: J. Infect. Dis. **59**:50, 1936. Werkman, C. H.; Baldwin, F. M., and Nelson, V. E.: ibid. **35**:549, 1924. Nedzel, A. J.: J. Lab. & Clin. Med. **20**:944, 1935. Wien, R.: Quart. J. Pharm. & Pharmacol. **9**:48 (Jan.-March) 1936. Cutler, J. T.: J. Pharmacol. & Exper. Therap. **45**:209, 1932.

patients have been given an excellent diet supplemented by certain vitamin products and allowed to continue their usual intake of tobacco and alcohol and that the results have been very satisfactory.

I wish to state that I have not recommended continuing the use of tobacco and alcohol in the ordinary case. The patients in this series have all been hospitalized and observed closely over a long period. There are other patients who have refused to abstain from alcohol and tobacco and who have taken the prescribed quantity of yeast at home. Thus far they have made satisfactory improvement, but it is too early to more than mention this group. This problem is still in the stage of clinical research, but I have no hesitancy in concluding that in the etiology and treatment of this condition diet does play an important role.

REPORT OF THE OTHER CASES

CASE 1.—C. S., a 28 year old Negro woman, who was mentioned previously,[15] was first examined in the Vanderbilt Clinic in October 1934. She had marked tremor of her tongue and fingers, and her breath had a strong alcoholic odor. The vision was 20/200 in each eye and was unimproved with glasses. The disks showed very marked temporal pallor consistent with atrophy of the papillomacular bundle, and the fields revealed depression of central vision. She had never used tobacco in any form but consumed between a pint and a quart of whisky daily. In three months vision improved to 20/70 in each eye and remained stationary. She was strongly advised to discontinue the use of alcohol, but she refused. Nine months after coming under observation she was admitted to the Institute of Ophthalmology. The disks appeared atrophic temporally. She had alcoholic gastritis, and two gastric analyses showed absence of free hydrochloric acid. During thirty-six days in the hospital she drank between 1 pint and 1 quart of liquor daily. (The liquor given to her and to all other patients and referred to hereafter merely as "liquor" consisted of equal parts of 95 per cent ethyl alcohol and water flavored with the juices from lemon and orange peels. It had a pleasant odor and taste.) She took the prescribed diet. After this period she felt that her vision had definitely improved. I was certain that her eyes were not worse. She could read several lines lower on the test chart than on admission, but I felt that her condition should be considered unimproved. She has been followed twenty-two months since discharge from the hospital, and her eyes have shown no further change. The diagnosis was alcoholic amblyopia and partial optic atrophy, unimproved.

CASE 2.—G. J., a 31 year old actor, a private patient of Dr. Gordon Bruce, entered the Institute of Ophthalmology on Dec. 3, 1935. He drank about 1 quart of gin and smoked from one to two packages of cigarets daily. Vision was 20/200 in each eye. Each disk was orange except temporally, where it was somewhat pale. The margins were indistinct, except temporally, and slightly elevated (less than 1 D.). Elevation of the disk is unusual in this condition. The fields showed typical centrocecal scotomas. Neurologic examination, roentgenograms of the head and the Wassermann test gave negative results. A medical consultant

15. Carroll, F. D., and Franklin, C. R.: Am. J. Ophth. **19**:1070, 1936.

diagnosed alcoholic gastritis and hepatitis. In the hospital the patient took from 1 pint to 1 quart of liquor daily, smoked from one to two packages of cigarets daily and faithfully ate what was given to him. In two weeks vision was 20/120 in the right eye and 20/50 in the left, and the margins of the disk appeared less elevated. In one month vision was 20/40 in the right eye and 20/30 in the left, and the scotomas had greatly decreased in size. The patient was discharged from the hospital on Jan. 24, 1936, and three months later vision was 20/30 in the right eye and 20/20 in the left. He has not abstained from alcohol or tobacco, but he stated in a recent communication that his vision has remained the same.

Case 3.—N. M.,[11] a 36 year old janitor, drank· about 1 quart of liquor and smoked one package of cigarets daily. He was first seen in the Bellevue Psychiatric Hospital on Dec. 12, 1935. He had pellagra, polyneuritis and alcohol amblyopia. Vision was 10/200 in the right eye and 8/200 in the left. Each disk showed marked temporal pallor compatible with optic atrophy, and the fields had centrocecal scotomas. When his condition permitted he was transferred to the Institute of Ophthalmology. Retinoscopic examination indicated a large astigmatic error in the left eye, and he stated that vision had never been good in that eye. For the first month in the hospital he was forced to abstain from all alcohol and tobacco, and there was slight improvement. Visual acuity increased from 18/200 in the right eye and 12/200 in the left eye to 20/120 in each eye with correction. He was then given from 1 pint to 1 quart of liquor daily and cigarets. He faithfully took his diet. In one month on this regimen vision increased to 20/50 in the right eye and to 20/120 in the left and the fields were improved, and in another five weeks, when he was discharged, vision was 20/40 in the right eye and 20/80 in the left.

Case 4.—W. F., a 49 year old Jewish waiter, had noticed decreased vision for four months. He had been to two ophthalmic clinics and when first seen by me was at the Neurological Institute, where he had been sent because of a suspected tumor of the brain. He smoked from ten to thirteen cigars daily and drank an average of two or three glasses of beer and an ounce (30 cc.) of whisky daily. Vision was 20/200 in each eye; the disks were normal in appearance, and the fields showed characteristic centrocecal scotomas (fig. 2 *A*). He was transferred to the Institute of Ophthalmology on May 13, 1936, and placed on the prescribed diet. He continued to smoke from nine to fifteen cigars daily and was given 6 ounces (177 cc.) of liquor daily. Neurologic examination showed some increase in the deep reflexes on the left. An otologic check-up revealed early toxic neuritis of both eighth nerves, and the audiograms were similar to those previously reported.[16] The diameter of the right pupil was 1 mm. larger than that of the left; there were small opacities in both the cornea and the lens of each eye. In two months vision was 20/50 — in the right eye and 20/60 in the left. In the next two months visual acuity did not improve, but the patient reduced his consumption of alcohol and tobacco somewhat. In three and a half months vision was 20/30 in each eye and the fields were as shown in fig. 2 *B*.

Case 5.—E. F., a 32 year old Negro postoffice clerk, who was previously mentioned,[11] had the alcoholic type of pellagra, polyneuritis and amblyopia. When

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16. Carroll, F. D., and Ireland, P. E.: Association of Toxic Deafness with Toxic Amblyopia Due to Tobacco and Alcohol, Arch. Otolaryng. **21**:459 (April) 1935.

he entered the Institute of Ophthalmology on May 14, 1935, vision was 3/200 in each eye (fig. 3 *A*). He abstained from alcohol and took all the prescribed food for thirty-five days. Vision by then had improved to 20/40 + in each eye, and he was discharged. He immediately started to drink and smoke as much as previously but regularly took the powdered brewers' yeast and wheat germ as prescribed. Vision gradually improved to 20/20 in each eye, and the fields showed
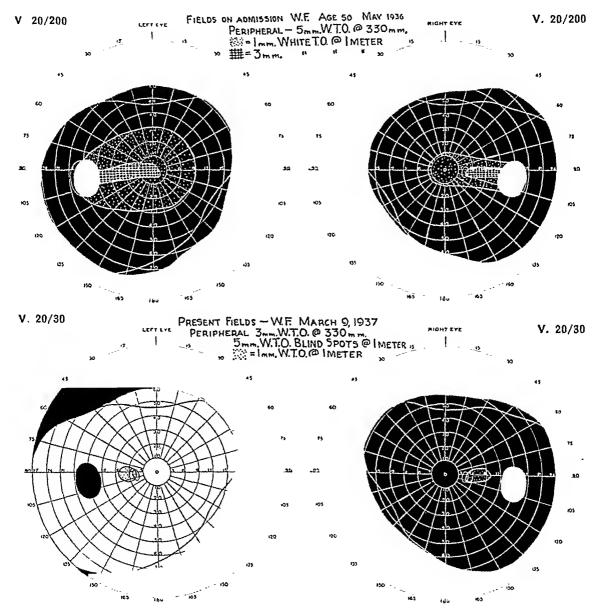
Fig. 2 (case 4).—Fields of W. F.

only a small indefinite scotoma between the blindspot and the fixation point. Then he stopped taking yeast but continued his usual intake of whisky (from 1 pint to 1 quart daily). In about five weeks the pellagra returned, and he came back to the clinic. He had mild typical pellagral dermatitis, pain in his legs and feet and diarrhea. His vision was still excellent. He was given more yeast. Then he disappeared until Dec. 17, 1936, when he came back so weak that he could walk only with difficulty. He had the dermatitis, stomatitis, diarrhea and

peripheral neuritis which accompany pellagra. He had continued his intake of alcohol, but instead of taking 4 tablespoonfuls of powdered brewers' yeast daily he had been using only a few tablets of powdered brewers' yeast daily. Vision remained 20/20 in each eye, and there was only the same small residual scotoma between the blindspot and the fixation area in each eye (fig. 3 *B*). He was again hospitalized and given powdered brewers' yeast in doses of 2 tablespoonfuls
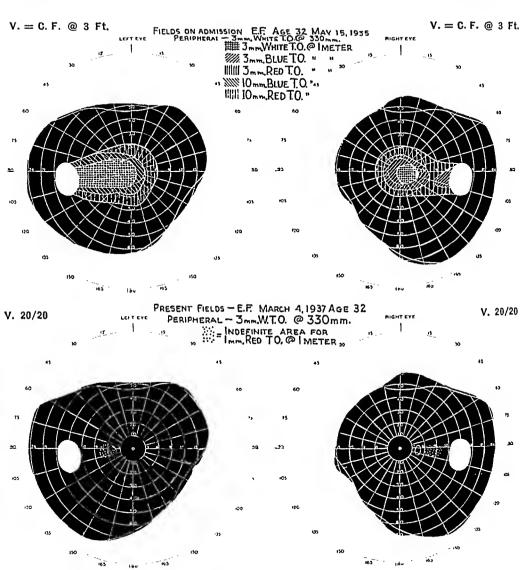
Fig. 3 (case 5).—Fields of E. F.

five times daily (90 Gm.), wheat germ in doses of 4 tablespoonfuls three times daily, vegex in doses of 1 teaspoonful three times daily and 5 or 10 cc. of liver extract [9b] intramuscularly daily. He was also given 1 pint of liquor daily. Gastric analysis showed lack of free hydrochloric acid. His condition again rapidly improved, and he was discharged in four weeks free from any symptoms. Since then he has continued his usual consumption of tobacco and alcohol but has faithfully taken 2 tablespoonfuls of yeast daily. He has no symptoms.

CASE 6.—H. R., a 63 year old man, was first seen in the Vanderbilt Clinic on May 29, 1935. His vision at that time was 20/200 in each eye with correction. The right eye had not had good vision since having a corneal ulcer twelve years previously. It showed diffuse corneal opacities and an incipient cataract. The left eye also had an incipient cataract, which was not sufficiently dense to explain the decreased vision. The disks appeared normal, and the fields showed typical
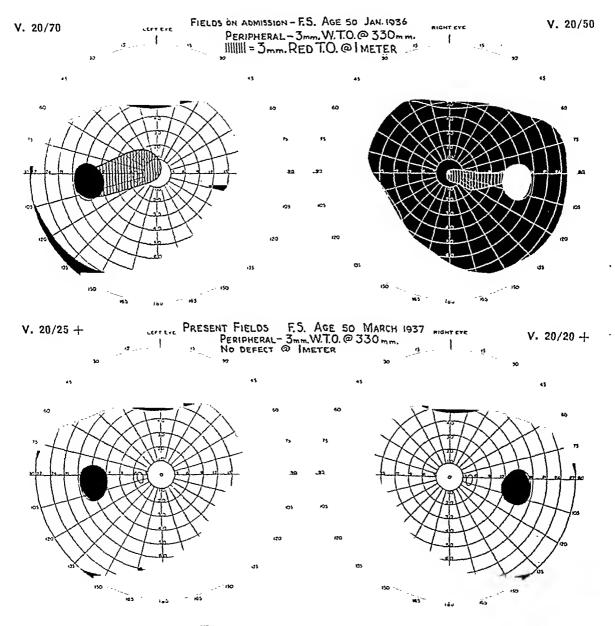
Fig. 4 (case 7).—Fields of F. S.

centrocecal scotomas. The patient drank four glasses of beer and smoked from ten to twelve cigars daily. He was advised to discontinue tobacco and alcohol and did so, but when I first saw him two months later, on July 26, vision had decreased to 10/200 in the right eye and to 5/200 in the left. He was admitted to the Institute of Ophthalmology and allowed to drink and smoke as much as he wished—this was about ten cigars daily and from 6 to 14 ounces (177 to

414 cc.) of liquor. He took all the prescribed diet. No improvement was seen for almost a month; then it came gradually. In two months vision in the left eye had improved from 5/200 to 20/80. The opacities in the cornea and lens of the right eye appeared sufficient to explain the poor vision of 10/200 in that eye. In three months vision of the left eye was 20/60 —, and on discharge from the hospital vision of the right eye was 10/200 and that of the left eye 20/40. There was marked decrease in the density of the scotomas. This patient remained in the hospital under close observation for a total of five months. During this time vision of the left eye, which was the only eye in which vision could be expected to improve, increased from 5/200 to 20/40. Except for one week, during this period he smoked an average of ten cigars daily and drank from 6 to 14 ounces of liquor daily. On discharge from the hospital he was advised to decrease or discontinue the use of tobacco and alcohol. It is not certain whether he followed this advice. Fourteen months since discharge vision of the left eye is the same—20/40+.

CASE 7.—F. S., a 50 year old unemployed Italian bank clerk, was referred to me for treatment by Dr. Charles Perera, who made the diagnosis of toxic amblyopia. Vision corrected was 20/50 in the right eye and 20/70— in the left; typical centrocecal scotomas were present (fig. 4 B). The disks were normal in appearance. The patient stated that his vision had been gradually decreasing for the past year. He was accustomed to drinking between 1 pint and 1 quart of wine and smoking one package of cigarets daily. For the past several years he had noticed some abdominal discomfort and during the past year had lost from 10 to 15 pounds (4.5 to 6.8 Kg.) in weight. His appetite was poor. On Jan. 20, 1936, he entered the Institute of Ophthalmology. He brought his own wine and cigarets with him, and throughout his stay in the hospital he used the same amount of wine and smoked the same number of cigarets that he had consumed while the tobacco-alcohol amblyopia was developing. He remained in the hospital under close observation for seven weeks. He took the prescribed diet. His weight increased 15 pounds (6.8 Kg.). His vision gradually improved to 20/20— in the right eye and to 20/30— in the left in seventeen days and was 20/20+ in the right eye and 20/30 + in the left when he left the hospital. Since then fourteen months have passed. At no time during this long course of observation has the consumption of alcohol or tobacco ever been reduced. He has continued to take 1 or two tablespoonfuls of yeast daily because I was not sure whether or not it would be safe for him to discontinue it. Vision now is 20/20+ in the right eye and 20/25+ in the left. I am unable to plot any scotoma in either eye (fig. 4 B).

ABSTRACT OF DISCUSSION

DR. ARTHUR M. YUDKIN, New Haven, Conn.: It is noteworthy that two distinct types of therapeutic measures should be offered at this meeting for the cure of the same ocular disturbance. Tobacco-alcohol amblyopia has been cured by other methods. One, in particular, is to eliminate the consumption of alcohol and tobacco and give the patient a well balanced diet. This method does not restore vision until the patient has adhered to the regulations for many months. At the Milwaukee session of the American Medical Association in 1933 I recommended the treatment of this disease on the basis of a well balanced diet supplemented by cod liver oil, and in some instances vitamin B complex was also given. Most of the patients improved, even though they consumed a moderate amount of alcohol and tobacco. When destruction of the

optic nerve was present, it was difficult to restore vision. Frequently a constitutional disturbance, such as diabetes, delayed the healing process. Dr. Carroll has presented a similar observation. In spite of his ability to cure this condition while the patient consumes his regular amount of alcohol and tobacco, Dr. Carroll realizes the advantages of diminishing and even eliminating the drugs.

From a scientific standpoint this paper is a valuable contribution, for it demonstrates that alcohol and tobacco when consumed by a person having a lowered resistance may produce not only a general condition but also ocular defects. The severity of the clinical picture depends on the general health of the patient and the amount of alcohol or tobacco consumed. Many cases are recorded of the appearance of vitamin B complex deficiency associated with voluntary restriction of food as a result of digestive disturbance, or as a therapeutic measure, as in diabetes, in pregnancy attended by vomiting, in alcoholism and in other conditions. Strange as it may seem, many deficiency diseases are now cured by the addition of large doses of vitamin B complex to the diet. Nevertheless it cannot be said that alcohol-tobacco amblyopia is due to vitamin B complex deficiency.

From experimental work, it can readily be seen why one type of symptoms appears in one group of animals and another type in still another. Dr. Cowgill and his collaborators kept one group of dogs on a minimum amount of vitamin B_2 (G), while another group was deprived of B_2 (G) entirely. The first group showed collapse, whereas the latter group suffered eventually from ataxia. Two quite different syndromes were observed. The picture of complete deprivation was different from that of what might be called subnutrition. That is probably why in one group beriberi appears, in another pellagra, and in still another ocular disturbance. It becomes evident that a person suffering from a deficiency may at some time or other have different nerves involved. In dealing with man it must not be forgotten that the intestinal tract plays a great role in the distribution of materials necessary for the upkeep of the body. The strains of modern life have produced certain nervous manifestations that have actually upset the whole gastro-intestinal mechanism, with the result that the food is not properly prepared for digestion and absorption. I recommend that the ophthalmologist not only treat his patients with local medication but become interested in the general health of his patient. If he is confident of his medical judgment, there is no reason why he should not advise the patient concerning diet and the mode of living.

In spite of the dramatic results obtained in tobacco-alcohol amblyopia by Dr. Carroll, I cannot subscribe to the continued use of these drugs except in exceptional cases, nor can I see the reason for such large amounts of vitamin B complex except for investigative purposes. Excellent results may be obtained in this ocular disturbance, which, I believe, is associated with a dietary deficiency probably of a gastro-intestinal nature, by the restriction of alcohol and tobacco, by the elimination of constitutional complications and by prescribing a sensible, well balanced diet supplemented with moderate doses of standardized brewers' yeast and properly assayed cod liver oil.

Dr. Frank Carroll, New York: The idea of Dr. Duggan that tobacco amblyopia is caused by a spasm of vessels supplying the optic pathway deserves critical consideration. There is some evidence that smoking constricts the peripheral vessels. A report published recently, however, indicates that there is no more peripheral vasoconstriction from smoking than there is from taking a deep inspiration of pure air. Several of my patients with toxic amblyopia who were heavy smokers have been tested by members of the department of pharmacology of Columbia University, and no evidence was found by an accurate plethysmograph method of any peripheral vasoconstriction. But even if smoking does tend to constrict the peripheral vessels and raise the blood pressure, this does not indicate that either the cerebral or the retinal vessels are constricted. In fact, just the reverse is apt to be true. For example, when the epinephrine is given, vasoconstriction of the peripheral vessels occurs. This causes an increased blood pressure with resulting vasodilatation of the cerebral vessels. Lambert has shown that in animals epinephrine, which causes constriction of peripheral blood vessels, tends to cause vasodilatation of the retinal vessels because of the increase in the blood pressure. If there should be a spasm, where would it be and what would be its nature? It is almost certainly not in the retina; the retinal vessels appear perfectly normal in many of these patients. The pathologic change is essentially limited to the papillomacular bundles, and therefore this hypothetic spasm would have to be limited to the vessels supplying the papillomacular bundle. The chief vessel supplying the papillomacular bundle is the central retinal artery, and when there is an ordinary spasm of this vessel it can be seen with an ophthalmoscope. It does not resemble anything seen in tobacco amblyopia. Moreover, this type of amblyopia comes on gradually and disappears gradually. Spasms of the central retinal artery or its branches that are familiar come on suddenly; therefore, one is expected to assume a special kind of spasm foreign to the present knowledge.

Dr. Duggan has stated that in tobacco-alcohol amblyopia moderate drinkers improve more rapidly than nondrinkers, and he thinks this is interesting in view of the vasodilating property of alcohol. How would he explain the existence of a disease, indistinguishable from tobacco-alcohol amblyopia in patients who smoke little or not at all, but who are heavy drinkers? Certainly alcohol is not a vasoconstrictor. According to this vascular theory, my patients with this disease who were heavy drinkers would have been especially immune because they were heavy drinkers and should never have had amblyopia—but they did. Vasodilators are suggested as being beneficial. One would expect a vasoconstrictor such as epinephrine to be of more value. This would raise the blood pressure and thus dilate the cerebral and retinal vessels as well as increase the rate of blood flow through the brain. Dr. Duggan has stated that the speed of recovery of patients receiving vasodilators is more rapid than that of patients not receiving them. This is a difficult point to prove and needs controls. An attempt has been made to use other cases reported in the literature as controls, but they are not comparable. If Dr. Duggan is to persist in his efforts to

prove the value of these drugs, he should treat every alternate patient with an equal number of injections of saline solution or even vaso-constrictors.

I have the records of nine patients with this condition who were treated with intravenous injections of sodium nitrite either by other members of the hospital staff or by myself during the past five years. An effort was made to give the vasodilator exactly as suggested by Dr. Duggan. Some patients received daily injections; others received injections twice daily. The average number of injections received by each patient was fifty. Two of the nine patients showed no improvement. The others gradually became better. I am sorry to say that there was nothing to indicate that the vasodilator had hastened the rate of recovery.

Dr. Yudkin suggested that perhaps I was using larger doses of brewers' yeast than necessary, and that may possibly be true. I do believe that large doses are essential if the patient continues to drink and smoke. One of my patients stopped taking what I suggested and took yeast cakes instead, and pellagra developed within a month. He then took the powdered brewers' yeast again, and the pellagra cleared up. He then failed to follow my advice and took tablets of brewers' yeast instead of taking powdered yeast in tablespoonful doses, and the pellagra returned. It rapidly cleared up again on the suggested dosage.

Dr. Walter F. Duggan, New York: I have read most of the reports that Dr. Carroll has read concerning the vasoconstrictive effect of smoking on the peripheral vessels and am amazed at the conflicting statements. I have treated with vasodilators alone some fifty patients with tobacco amblyopia without atrophy of the optic nerve. The visual improvement has been so rapid in many cases that I have been surprised. Dr. Fralich and Dr. Bracken, of New York, have obtained good results. I have only the improvement in vision, the patient's word and the decrease in the size of the scotoma on which to base my figures. As to where the spasm is located, Dr. Carroll mentioned that the papillo-macular bundle is involved and that it is supplied by the central artery of the retina. It is, in the retina, but the central artery of the retina usually does not supply any fibers of the optic nerve posterior to the point at which it enters the optic nerve, and it certainly does not supply the papillomacular bundle in the chiasm. Behr recently mentioned that the central artery of the retina is strictly a retinal artery as soon as it enters the optic nerve. My feeling is that the arterioles which are involved are in the region of the chiasm, because of the bilateral field defects. I have seen no change, as a rule, in the retinal vessels after this treatment, but I have seen an improvement in vision and a decrease in the size of the scotoma. Tobacco amblyopia does not come on suddenly. Amblyopia does come on suddenly in cases of retrobulbar neuritis, and I feel that this is much more analogous to a spasm of the central retinal artery than toxic amblyopia is. As to why these particular vessels (in the chiasm?) are involved, I have no explanation other than that they are end-arterioles. I think that Dr. Carroll's cases

and my cases belong to different groups. In the nine cases that he reported in December 1936 the average age was 36. The average age which has been found by all authors in cases of tobacco amblyopia is between 50 and 56 years. In cases of real tobacco amblyopia the patient does not show polyneuritis or symptoms of pellagra. In the cases which Dr. Carroll reported the patients did show symptoms of a deficiency disease. I think that he and I are discussing different disease entities.

INTRA-OCULAR INVASION BY THE LARVA OF THE ASCARIS

REPORT OF A CASE WITH UNUSUAL COMPLICATIONS

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ATLANTA, GA.

It is a rare occurrence for the interior of the eye to be invaded by any parasite. Of those parasites classified as the nematodes, the filaria has been found within the eye by Wright and his collaborators,[1] Fernando[2] and others, and Fulleborn[3] stated the belief that the retinal hemorrhages seen in hookworm infections are the results of embolic embryonic larvae of the uncinaria.

The ascaris (Ascaris lumbricoides) is one of the most common of the nematoda, and Ward[4] commented in his monograph on ocular parasites that it is only indirectly parasitic to the eye, such as through some constitutional ailment. However, this intestinal round-worm (Ascaris) has been found in its young or adult form in many organs of the human body, even in the brain and in the heart,[5] and it seems reasonable to believe that through the same channels, the circulation or the lymphatics, it might gain access to the interior of the eye.

1. Wright, R. E.: Adult Filaria (Wuchereria Bancrofti) in Anterior Chamber, Brit. J. Ophth. **18**:646, 1934. Wright, R. E.; Iyer, P. V. S., and Pandit, C. G.: Description of an Adult Filaria (Male) Removed from the Anterior Chamber of the Eye of Man, Indian J. M. Research **23**:199, 1935.

2. Fernando, S. E.: Ocular Filariasis (Adult *Wuchereria bancrofti*) in the Anterior Chamber of Human Eye, J. Trop. Med. **38**:17, 1935.

3. Fulleborn, F.: Untersuchungen über den Infektionsweg bei Strongyloides und Ankylostomum und die Biologie dieser Parasiten, Arch. f. Schiffs- u. Tropen-Hyg. (supp.) **18**:182, 1914.

4. Ward, H. B.: Ocular Parasites, in Wood, Casey A.: American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1918, vol. 12, p. 9299.

5. Boettiger, C., and Werne, J.: Ascaris Lumbricoides Found in the Cavity of the Human Heart, J. A. M. A. **93**:32 (July 6) 1929. Sterling, R., and Guay, A. J. L.: Invasion of the Female Generative Tract by Ascaris Lumbricoides, ibid. **107**:2046 (Dec. 19) 1936.

The experimental work of Suyemori [6] supports this belief, for after feeding guinea-pigs the eggs containing fully developed embryos, "ascaris larva in the ocular organs were seen moving with blood—30 hours after infection," and were later found in the retina and optic nerve and in abundance in the uveal tract.

The case to be reported is one of intra-ocular invasion by a larva. Its position on the lens offered an excellent opportunity for study with the slit lamp and the microscope, and it was classified as the larva of an ascaris. I am conscious that for scientific accuracy this opinion might be challenged, as the larva could not be removed for dissection and microscopic study, yet the evidence is so strongly in favor of it being the larva of an ascaris that this unique case is reported.

REPORT OF CASE

History.—On Dec. 1, 1936, J. Y., aged 8, was referred by a practitioner in a rural community of Northern Georgia, the opinion being offered that the patient had acute glaucoma. The eye had been inflamed and painful for three days; there was no history of trauma, and the father reported that so far as he knew the boy was healthy. The patient had never been outside of his county. The father, a fairly intelligent farmer, gave a good account of himself and his large family. The only information which was of value (then it was thought to be inconsequential) was that two other children had been recently treated for "worms."

Ocular Examination.—The right eye had vision permitting perception of movements of the hand. The globe was intensely injected. The cornea was steamy, with several keratitis dots on the posterior surface; the anterior chamber was deep, and the aqueous was cloudy. The iris was off color; the pupil was irregular, semidilated and blocked by a thin exudate which prevented an ophthalmoscopic examination. The intra-ocular tension was 37 mm. of mercury (Schiötz). The external and the internal examination of the left eye gave normal results.

An opinion was expressed that the patient had acute iridocyclitis associated with secondary glaucoma of undetermined origin, and as the father would not consent to his son remaining, drops of atropine sulfate and hot packs were ordered, with the caution that the home physician should closely watch the eye.

One week later the eye was moderately injected; the pupil was widely dilated except for dense adhesions at 9 o'clock, and only a fundus reflex was obtained. The tension by palpation was normal.

In two weeks the eye was comfortable and rapidly clearing. The pupillary area was now clear except for the adhesions at 9 o'clock and some scattered pigment on the anterior surface of the lens. The lens was found to be dislocated temporarily, and in the adjacent circumlental space there were exudates which extended into the vitreous. The tension was plus one.

I suspected that the eye had been traumatized, but the patient and his father were equally positive that such was not the case.

In the fourth week the patient was still comfortable, and the globe was only slightly injected. The tension was plus one. The cornea was faintly edematous;

6. Suyemori, S.: On the Pathological Changes in the Eye During the Incipient State of Infection with Ascaris (Experimental Study), Taiwan Igakkai Zasshi, 1925, vol. 1, no. 239.

the iris was dilated and tremulous, and the exudates in the circumlental space had increased. Coming from behind the iris at 5 o'clock and lying on the anterior surface of the lens was seen for the first time a slender, translucent pearly white object, which had the appearance of a thin delicate spear point, for the sides were beveled and a distinct central line coursed down from its rounded end to where
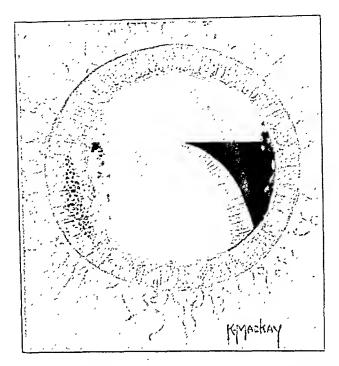
Fig. 1.—Larva of Ascaris on the anterior surface of the lens.
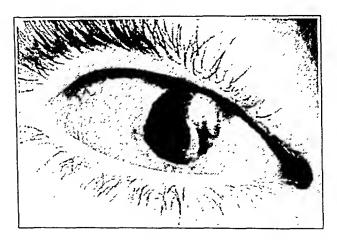
Fig. 2.—Unretouched photograph of the larva of Ascaris.

the object passed behind the iris. The object was nonmotile, had a slight slant temporalward and measured 3 mm. long, and at the pupillary margin its maximum width was 1 mm. It was suggested that this object was the larva of an intestinal parasite. The following day several stools were examined, but no ova were found.

In the fifth week the eye was very quiet, and the tension was normal. The larva had decidedly grown and measured 1.5 mm. by 5 mm. It had assumed a distinctly curved position and was now definitely round. Throughout its entire

length were delicate horizontal lines, and running down its center was a brownish marking. When the globe was rotated downward one could readily see with the ophthalmoscope that the larva curved around the equator of the dislocated lens and extended 2 mm. beyond; it was attached to the posterior surface by its head. The fundus could now be seen for the first time, and it was found to be normal. The opacities in the vitreous were entirely confined to the lower nasal sector.

Dr. Thomas F. Sellers, parasitologist of the Georgia State Board of Health, was asked to see the eye, and he expressed the opinion, after making several slit lamp examinations, that the larva had all the morphologic characteristics of an immature ascaris.

The patient was hospitalized for a complete study; the results of all the examinations were normal. Repeated examinations of the stools showed no ova of intestinal parasites. Examination of the blood showed 75 per cent hemoglobin, 4,340,000 red cells, 8,200 white cells, 51 polymorphonuclears, 47 lymphocytes and 2 eosinophils. Smears gave negative results.

With the knowledge that the cuticle and celomic fluid of the ascaris is often toxic and as the eye was becoming more quiet, it was decided that the best policy would be to wait for developments, in the hope that the larva might find its way into the anterior chamber, from which it could be more readily removed. Treatment with drops of atropine sulfate and hot packs was continued.

At the patient's visit in the sixth week the eye was still quiet, and the tension was normal. The body of the larva appeared to be less glistening, and scattered over its surface and on the adjoining capsule of the lens were seen delicate brownish markings, which, when viewed with the slit lamp, resembled the curled-up bark of a silver birch tree; these were interpreted as being cast-off particles of desquamated cuticle. The size of the larva had not changed, and it was believed that it was disintegrating.

The eye remained quiet until the eleventh week, when the patient returned with the statement that it had been inflamed for the past four days but there had been no pain. The globe was quite congested, the aqueous cloudy, the iris off color, and the pupil slightly contracted, in spite of the daily use of atropine sulfate. The iris was adherent to the larva on the nasal side, and the detritus coming from the desquamated cuticle had increased.

The tension of the right eye was 10 mm. of mercury, and that of the left eye was 17 mm.

It was now thought that the acute flare-up of iritis was due to the disintegration of the larva and that the best procedure would be to remove it.

Operation.—With the patient under induced anesthesia with ether, a limbal incision was made with a thin knife from 4 to 7 o'clock. This approach was decided on for the reason that if the texture of the larva was reasonably tough, as is that of the adult worm, it could be grasped and teased out without further dislocation of the lens or loss of vitreous.

An attempt was made to grasp the larva with a capsule forceps, but, to my astonishment, its substance was like jelly; only fragments could be removed, and there remained the tail, delicate fragments on the surface of the lens, the part behind the iris and the head. The anterior chamber was gently irrigated with saline solution, in the hope that these particles might flow out, but they were firmly adherent to the lens. As the iris tended to prolapse, a drop of solution of

physostigmine was used and a firm bandage applied. At the first dressing the globe was found to be quiet, but the iris had prolapsed. This was excised, and the pillars were replaced.

Outcome.—The eye made a prompt recovery, and when the patient was last seen, on June 27, seven months after the onset, vision was 10/200, and the tension was normal. The pupil was dilated, and there was a small keyhole-shaped coloboma of the iris at 5 o'clock. The old adhesion of the iris at 9 o'clock was still present, and on the anterior capsule were a few deposits of pigment. No trace of the larva could be seen on the anterior or the posterior surface of the lens. Several fine lines were seen in the cortex of the dislocated lens, and a few vitreous opacities remained.

COMMENT

In the case which is presented every symptom is referred to the affected eye, and the evidence is that the iridocyclitis and even the dislocation of the lens were the direct results of intra-ocular invasion by a parasite, which was classified as the larva of an ascaris.

The mode of infection and the cycle of development of the ascaris may be recalled with some interest. The ova are carried by contaminated food into the intestinal tract, where the larva are liberated by the hatching of the eggs in the small intestine and then penetrate the portal circulation. After a brief stay in the liver they pass through the right side of the heart into the lung and undergo further development in the capillaries; thence they pass into the vesicles, up the trachea, and down the esophagus into the stomach and intestine, where they develop into adult worms.

At the outset one must assume that the patient had a mild parasitic infection, notwithstanding the fact that no ova were found in the stools. The fact that stools contain no ova does not always exclude parasitic infection. It is known that the patient was exposed to the same home influences which caused two other children in the family to be treated for "worms."

The fact that no other case of intra-ocular invasion by the larva of an ascaris has been reported should not deter one in the belief that it is impossible. On the contrary, such an invasion would seem most logical, since the ascaris has been found in other organs of the body, notably, the heart and brain. It is difficult to say whether this living embolus passes through the lymphatics or the arterial circulation, although it is possible to conceive that an embryonic larva while in the lungs during the period of migration could pass through the pulmonary capillaries into the general circulation and, in cases in which the eye is involved, gain entrance through any penetrating vessel.

Since the iris and ciliary body were early involved in this case, one may assume that an anterior ciliary artery, located approximately

at 5 o'clock and supplying those structures, was the carrier. However, the larva might have entered at 9 o'clock, the site of the dense adhesion of the iris, left its infection and migrated. It finally attached itself by the head to the posterior surface of the lens and extended its caudal end into the proximal fibers of the suspensory ligament, which were likely torn or worn away for a considerable extent by its motility and growth. The destruction of the nasal zonular fibers, combined with the traction of the dense adhesions in the temporal portion of the iris, offers a reasonable explanation for the temporal displacement of the lens noted at the third visit, one week before the larva made its appearance. Indeed, one may speculate further and suggest that this dislocation may have been one reason why the infection was largely limited to the iris, as it allowed the larva to escape forward through the large circumlental space instead of being confined to the ciliary region.

It is commonly known among parasitologists that the cuticle and the celomic fluid of the ascaris (especially Ascaris megalocephalia) are often toxic, and it was thought that any attempt to remove the larva during the period of the acute ocular infection would have added injury to the globe. For that reason and because the inflammatory symptoms were rapidly subsiding, it was deemed best to postpone any operative procedure, in the hope that the larva might free itself and fall into the anterior chamber.

In the fifth week it was noted that the larva was dying and undergoing degeneration, as evidenced by lack of motility, no increase in size and especially desquamation of the cuticle. A week later there was a recurrence of the iritis, and it was then thought that the globe could best be preserved by removal of the larva. What happened at the operation is recalled in the report of the case.

The fact that the eye showed no reaction after the operation, even when fragments of the larva remained, now leads me to believe that the wisest procedure would have been not to disturb it and that in time the larva would have been completely absorbed. There is no precedent for this opinion, except in Anderson's [7] report of a case of ophthalmomyiasis interna, in which the larva of an insect having migrated into the vitreous, absorbed after eighteen months, with no injury to the eye.

In attempting a differentiation of the types of larva which would present pictures resembling the one in this case, two possibilities can be considered; the larva of the fly and the larva of a nematode.

The former type should be ruled out, as this larva is always segmented and its surface in no way resembles the round, smooth surface

7. Anderson, W. Banks: Ophthalmomyiasis, Am. J. Ophth. **18**:699, 1935.

of the worm that has been described. Besides there is often the history of the insect striking the eye and becoming lodged in the fornix, with the usual discomfort, which was not present in this case.

In regard to the latter type, I present the report of Dr. Thomas F. Sellers, parasitologist of the Georgia State Board of Health, who aided in the preparation of this paper.

"The morphologic features of the foreign body, as described by Dr. Calhoun, were characteristic of the caudal end of a larva of a nematode. The tapering contour ending in a rather sharp point, the smooth cuticle showing only faint transverse striations, the slender lightly pigmented central canal—all these characters were present.

"The common nematodes parasitizing man are: the hookworm (Uncinaria americana and Ancylostoma duodenale), the pinworm (Oxyuris vermicularis), the whipworm (Trichuris trichiura), the round-worm, or the stomach worm (Ascaris lumbricoides), the trichina (Trichinella spiralis), Strongyloides stercoralis and several species of filaria. Of these, the filaridae, especially Filaria loa, have been known to parasitize the eye. That one was not dealing with the filaria in this case is evident, for none of the morphologic characters distinctive of the microfilaria were present, and in man this parasite is exceedingly rare in the interior of this country. The larva of the pinworm and that of the whipworm complete their life cycles from egg to adult entirely within the intestinal tract. They never invade the blood stream. The larva of the trichina does invade the blood stream and has been found encysted in the ocular musculature. This larva, however, is entirely too small to be considered in this case. The larva of Strongyloides likewise invades the blood stream, but it, too, is almost microscopic in size.

"This leaves for consideration only the hookworm and round-worm. The life cycles of these are somewhat similar. It is probable that some of these larvae, which are very minute, fail to escape to the air passages, and continue on into the pulmonary arterioles and thence back to the heart, to be distributed via the arterial circulation to any part of the body. Just how far such a larva thus entrapped could develop is not known. Nevertheless, one did develop in this case to such size as to protrude a good half its length around the lens and into the anterior chamber, so that 5 mm. of its length was plainly visible. The total length of the worm must have been at least 10 mm., perhaps 15 mm. Since this approximates the length of the young adult hookworm and yet this parasite had none of the characters of an adult form, it is believed that the hookworm can also be eliminated. Therefore, my opinion is that one was dealing with an entrapped larva of an ascarid, which, after progressing to a certain point in its unnatural environment, died and was in process of decomposition when Dr. Calhoun operated."

SUMMARY

The case of a young boy is presented, with an account of the following train of symptoms, recorded at weekly intervals, and my conclusions.

In the first week there were acute iridocyclitis and secondary glaucoma.

In the second week the eye was more quiet, and the tension was normal.

In the third week the lens was dislocated.

In the fourth week the appearance of a larva on the anterior surface of the lens was noted.

In the fifth week the larva had almost doubled its size.

In the sixth week there was evidence that the larva was dying and disintegrating.

In the eleventh week there was an acute flare-up of the iritis. The larva was definitely disintegrating, and an attempt to remove it was unsuccessful.

In the twelfth week the eye was quiet.

In the seventh month the eye was quiet. Complete absorption of the larva had occurred. The larva was identified (by T. F. S.) as an immature ascaris.

This experience now leads me to believe that the larva would have absorbed had it not been disturbed.

NEUROGENIC ORIGIN OF CHOROIDAL SARCOMA

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The main object of this paper is to show that the neoplasm of the choroid which has hitherto been described in the literature as choroidal sarcoma is much more probably a neurogenic tumor, arising from the Schwann sheath cells of nerves traversing the choroid. The neural origin of so-called sarcoma of the choroid will be supported by the histologic observations in seven cases which I have studied. The work, so far as I know, is new.

Before presenting and discussing my histologic studies, it is desirable to summarize briefly what the literature shows concerning neurogenic tumors in general, as well as to glance at the neurologic structure of the choroid and the neural and other neoplasms that are found in this vascular membrane.

NEUROGENIC NEOPLASMS IN GENERAL

Neoplasms showing more or less nerve tissue in their structure have been known for a long time. Tumors, especially those of the nervous system, composed of pure ganglion nerve cells and nerve fibers occurring in the course of a nerve have been called neuroma. Their existence has been denied by some, but Bruce and Dawson[1] reported them in scattered nerve tissue as embryonic rests.

More often tumors, single or multiple, containing nerve tissue have been found occurring along the course of nonmedullated, deep-lying nerves associated with fibrous tissue. Some show a preponderance of neural tissue and approach the neuroma type. Others are richly cellular, showing all kinds of spindle cells arranged in intertwining fascicles.

The cellular unit of the nervous system is the neuron, composed of a cell body, dendrites and a neurite or axon, which ends in teledendrons. Most of the nerves contain only neurites and no cell bodies, but some peripheral ganglionated plexuses of the sympathetic nervous system have nerve cells scattered among the nerve fibers. Peripheral nerves are medullated; deep-lying nerves are not. The neurite in medullated

1. Bruce, A., and Dawson, J. W.: Multiple Neuromata of the Central Nervous System, Rev. Neurol. & Psychiat. 11:235, 1913.

nerves is covered by myelin, a product of the nerve cell; outside of the myelin, which is absent in nonmedullated nerves, is the protoplasmic envelop, the sheath of Schwann, consisting of a syncytium of cells which migrate from the neural crest in early embryonic life. Some doubt has existed as to the nature of the sheath of Schwann. Harrison,[2] I believe, was the first to show its ectodermal origin. Penfield [3] described it mesodermal connective tissue. Recent studies, however, seem to have confirmed its ectodermal nature. The three structures of the peripheral nerve proper—the neurite, the myelin and the sheath of Schwann—are therefore taken here as ectodermal in origin. The whole nerve fiber is embedded in connective tissue, the endoneurium and perineurium, which are admittedly mesodermal. The pivotal importance of the ectodermal origin of the Schwann sheath cells to the histogenesis of neurogenic neoplasms will presently appear.

The uncertainty of pathologists regarding the nature of neurofibrous tumors is expressed in the variety of their names. They are known as Recklinghausen's fibromatosis, neurinoma, sympathetic blastoma, fibroma and neurofibrosarcoma and by other names. The tumors may arise along the course of either medullated or nonmedullated nerves.

Ewing [4] said that in actively growing neurosarcoma the cells are very numerous, are small, spindle shaped or round and are devoid of definite stroma. Kaufmann [5] stated that sarcoma composed largely of spindle cells may develop as the cell-rich malignant variety of neurofibroma from the connective tissue of nerves. Mallory [6] and other American pathologists agree that neural tumors may arise from the supporting endoneural connective tissue and therefore are mesodermal in origin. In the recent edition of his "Text-Book of Pathology" MacCallum,[7] admitting the ectodermal origin of the Schwann sheath cells, expressed the opinion that in proliferation these cells in neurinoma revert to an embryonic stage in their development, since they proceed to excessive growth which only passively includes nerve fibers.

2. Harrison, R.: Neuroblast Versus Sheath Cells in the Development of Peripheral Nerves, J. Comp. Neurol. **37:**123, 1924.

3. Penfield, W.: (*a*) Encapsulated Tumors of the Nervous System, Surg., Gynec. & Obst. **45:**178, 1927; (*b*) Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932.

4. Ewing, J.: Neoplasic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928, pp. 186, 466, 167 and 931.

5. Kaufmann, E.: Lehrbuch der speziellen pathologischen Anatomie, ed. 8, Berlin, W. de Gruyter & Co., 1922, p. 1566.

6. Mallory, F. B.: Type-Cell of the So-Called Dural Endothelioma, J. M. Research **41:**349, 1920.

7. MacCallum, W. G.: A Text-Book of Pathology, ed. 6, Philadelphia, W. B. Saunders Company, 1936, p. 1045.

The view that the neurofibrous tumors, especially neurosarcoma, arise from the supporting endoneural connective tissue, was generally held until 1910, when Verocay [8] published a report in which he showed clearly that they are derived from proliferation of Schwann sheath cells. Masson,[9] of Montreal, has published two important and convincing histologic studies in which he confirmed and extended Verocay's work, clearing up much misconception of neural neoplasms. In the first paper,[9a] published in 1926, he proved conclusively that pigmented cutaneous nevi are in reality neural tumors with preponderance of proliferation of Schwann sheath cells. Furthermore, he noted the Wagner-Meissner type of tactile corpuscles in schwannian tumors and concluded that the plexus of Schwann sheath cells which composes the greater part of nevi proceeds from sensory nerves. He contended further that in certain nevi the Schwann cells can fabricate melanin; judging by the behavior of melanoblasts in nevi, he attributed a neural nature to these pigmented cells, thus affording a point of view differing from that of the epithelial or connective tissue origin of melanoblasts in the skin.

I must pause here to remark somewhat on melanotic neoplasms generally. Melanotic sarcoma or melanoma was first described in 1784 as occurring in horses. Laënnec was the first to write on this type of tumor in man. Ever since, according to Coley and Hoguet,[10] discussion has been continuous as to the origin of melanotic tumors, the question being whether they arise from connective tissue or from epithelial tissue. Outside of the uveal tract they usually develop from congenital moles or pigmented warts. The majority of investigators at present agree with Masson and his followers that they are ectodermal in origin; however, Coley and Hoguet expressed the view that some melanotic tumors originate from connective tissue.

Nitsch,[11] of the Vienna Eye Cinic, said that pigmented tumors arise by preference from the sympathetic nerves. Parsons [12] observed that in the case of melanotic tumors there can be no doubt that the pigmented cells arise from the chromatophores, and he considered pigmented

8. Verocay, J.: Zur Kenntnis der Neurofibroma, Beitr. z. path. Anat. u. z. allg. Path. **48**:1, 1910.

9. Masson, P.: (*a*) Les naevi pigmentaires, tumeurs nerveuses, Ann. d'anat. path. **3**:417 and 657, 1926; (*b*) Experimental and Spontaneous Schwannomas, Am. J. Path. **8**:367, 1932.

10. Coley, W. B., and Hoguet, J. P.: Melanotic Cancer, with Report of Eighty Cases, Tr. Am. S. A. **34**:319, 1916.

11. Nitsch, Max: Neurofibromatose des Auges, Ztschr. f. Augenh. **69**:117 (Sept.) 1929.

12. Parsons, J. H.: Pathology of the Eye, London, Hodder & Stoughton, 1905, vol. 2, p. 518.

nevi as groups of densely packed chromatophores. Friedenwald[13] expressed a similar opinion.

Up to a few years ago most authors seemed to agree in admitting the connective tissue origin of chromatophores. Dr. Mary Knight,[14] in a study of melanotic neoplasms observed in the Mayo Clinic, expressed the opinion that melanin is produced only in ectodermal cells and that chromatophores are merely carriers of it. Broders and MacCarthy,[15] also of the Mayo Clinic, said positively that the pigment-bearing cells of the skin have their origin in ectoblastic rather than in mesoblastic tissue; they conceded that the origin of pigment cells in the eye is doubtful. None of these authors, however, has given any definite proof of his statements.

Regarding pigment in the uveal tract, distinction should be made between the construction of pigment and pigment-bearing cells. Miescher,[16] who did some excellent investigational work on this subject, found that pigment construction in the eye occurs only in the embryonic stage and that it is an oxidative process resulting from the action of an oxidative ferment, dopa-oxidase. The process of pigment construction is the same in the eye as in the skin. Proof of the presence of oxidase results from the dopa reaction,[17] which remains positive in pigment epithelium until the completion of pigmentation.

Oxidase occurs only in embryonic life; after the completion of pigment construction no more pigment is formed, and the cells concerned in pigment construction, whether melanoblasts or chromatophores, become, according to Miescher, only carriers of pigment during ordinary adult life. However, if there is any process of malignant degeneration the cells return to their original embryonic character and then have again the property of forming oxidase and resulting pigment. Under such circumstances, that is, in the presence of malignant degeneration in pigmented areas, the dopa reaction will again become positive. The results of the dopa reaction are, accordingly, an unmistakable proof that it is not a reaction of the pigment itself, as several authors have con-

13. Friedenwald, J. S.: Melanoma of the Choroid and Allied Tumors, in Penfield,[3b] vol. 3, p. 1065.

14. Knight, Mary S.: Melanotic Neoplasms of the Eye, J. A. M. A. **83**:1062 (Oct. 4) 1924.

15. Broders, A. C., and MacCarthy, W. C.: Melano-Epithelioma: Report of Seventy Cases, Surg., Gynec. & Obst. **23**:28, 1916.

16. Miescher, G.: Die Pigmentgenese im Auge nebst Bemerkungen über die Natur des Pigmentkorns, Arch. f. mikr. Anat. **97**:326, 1923.

17. Bloch, a distinguished dermatologist of Zurich, isolated a substance allied to tyrosine from the embryo of the broad bean. This substance (3, 4-dioxyphenylalanine) he called dopa. Its reaction is specific for all melanin-producing cells; these cells contain a ferment which converts dopa to melanin; the newly formed melanin colors the cell black.

sidered, but one that depends on the process of pigment building; the reaction takes place in the protoplasm of cells concerned in pigment construction.

Masson's [9a] assertion that in certain nevi the Schwann sheath cells can fabricate melanin has already been mentioned. In the histologic study of neurogenic pigmented choroidal tumors observed by them, Berger and Vaillancourt [18] extended Masson's conception; they noted that all or the greater part of the pigment cells in the eye as well as in the skin are of schwannian nature and that in neoplastic conditions melanogenesis can be assumed by neural elements, by the Schwann cells or by the syncytium of the Schwann sheath cells. This fact, according to these investigators, is absolutely incontestable. They observed that the tumor cells are capable of giving origin to both neural and pigment elements at once; these cells may differentiate on the one hand, as ganglion cells or typical Schwann cells, or, on the other hand, as pigmented cells free from all schwannian connections. This means, according to their theory, that pigment cells are nothing more than a particular differentiation of nerve elements; they also observed that this phenomenon of a double evolutive power of neoplastic cells in cases of melanotic tumors is not infrequent in cases of other neoplasms.

If these observations of Miescher,[16] Masson,[9] and Berger and Vaillancourt [18] regarding the origin of pigmented cells are correct, they will solve some of the puzzling questions that have arisen in the past regarding pigment and pigmented neoplasma. They are in harmony with the view of Knight [14] and others, who asserted that pigment is a product of ectodermal cells. Furthermore, Miescher's interesting disclosure that in the presence of malignant degeneration, sarcoma for instance, the normal cells revert to an embryonic state is in line with the view of Borst [19] and others, who have stated the opinion that sarcoma develops not from normal cells but from embryonal groups.

I must now return to the consideration of Masson's work concerning neurogenic neoplasma. In his second paper,[9b] published in 1932, he showed by histologic and biologic investigation of spontaneous and experimentally produced encapsulated nerves (including Recklinghausen's fibromatosis, schwannoma, neurinoma and all types of sensory and motor neuroma) that all such neural tumors have as their fundamental element proliferating cells of the nerve sheath of Schwann and are therefore ectodermal and do not originate from the mesodermal fibroblast. He showed that the collagen in these tumors is derived from the neurectoderm. He denied that the connective tissue of the endo-

18. Berger, L., and Vaillancourt: Ganglioneurome mélanique, Bull. Assoc. franç. p. l'étude du cancer **23**:275, 1934.

19. Borst, M.: Die Lehre von den Geschwülsten, Wiesbaden, J. F. Bergmann, 1902, p. 326.

neurium and perineurium has any part in the evolution of the neural tumor proper, though he stated that its proliferation may be stimulated by the adjacent tumor. Such growth of connective tissue may at times be so great as to stifle the Schwann cells. The syncytium of the Schwann sheath cells proliferates when from any cause the axon which it encloses has become separated from its trophic center and degenerates. Study of the syncytium of the Schwann sheath cells shows that they are capable under such circumstances of being transformed into bundles of aneuritic regenerated cells; when such bundles invade the ordinary connective tissue about them they are separated from it by endotheliform perineurium, formed in all probability not by the common connective tissue cells of the region but by cells emigrating from the endoneurium of the degenerated nerve.

The point is that a peripheral neural tumor develops independently of connective tissue, although proliferation of connective tissue in its vicinity may be stimulated. In cases in which the proliferation is extreme the Schwann sheath cells may be mistaken for connective tissue cells or for endoneural cells. However, Masson considered that there can be no doubt of their origin from neural elements.

In the United States Masson's histologic work has been confirmed by Foot,[20] Stewart and Copeland,[21] Geschickter [22] and others. Foot confirmed the schwannian origin of melanoma especially. Stewart and Copeland found that the fundamental cell of origin of so-called neurogenic sarcoma is the Schwann sheath cell. Geschickter showed that of nine hundred tumors of the peripheral nerves studied in the surgical pathologic laboratory of Johns Hopkins University eight hundred and fifty originated in the Schwann nerve sheath and that histologically there was a remarkable degree of uniformity in the majority of these tumors. He pointed out that proliferation of neurites does not take place unless Schwann sheath cells are present; such proliferation in the presence of ordinary connective tissue alone is highly hypothetic.

The work of the investigators cited has made it clear that the tumors described in the literature under the names of neurofibroma, plexiform or cirsoid neuroma, ganglionic neuroma, neurogenic sarcoma, nevi affecting nerves, melanoma and under other names are a closely related group allied to Recklinghausen's disease. They are neural neoplasms, the fundamental cell of origin of each being the Schwann sheath cell.

20. Foot, N. C.: Concerning Histology of Melanoma, Am. J. Path. **8**:321 and 619, 1932.

21. Stewart, F. W., and Copeland, M. W.: Neurogenic Sarcoma, Am. J. Cancer **15**:1235, 1931.

22. Geschickter, C. F.: Tumors of the Peripheral Nerves, Am. J. Cancer **25**:377, 1935.

The histologic similarity between the neural malignant tumors (especially so-called neurogenic sarcoma) and malignant tumors of connective tissue has been responsible for much confusion. Geschickter and Copeland [23] dwelt on the close resemblance that neurogenic tumors bear to ordinary spindle cell tumors. Like fibrosarcoma, the neural tumor may have a marked fibrillary structure and under the microscope may show many spindle cells. Also, like the fibrous spindle cell tumors, which show a continuous gradation from the benign fibroma to the most malignant spindle cell form, neural tumors show a gradual transition from benign neuroma to the most malignant form, called neurogenic sarcoma. The pathologic distinction between tumors of the spindle cell series and tumors of the neurogenic series is not made easily. However, the existence of neural tumors as a distinct class would appear to warrant a new classification not based on cellular characteristics.

With this new orientation of neural tumors in mind I shall now discuss neural tumors of the choroid. What I desire to establish is that tumors described up to this time in the literature as sarcoma of the choroid are either pigmented or unpigmented neural neoplasms originating from Schwann sheath cells of the nerves that traverse the choroid. It will be necessary, therefore, to review the histologic features of these nerves.

NEUROLOGIC STRUCTURE OF THE CHOROID

Minot [24] in his "Human Embryology" (1892) stated that the choroid is of ectodermal origin. Later writers, however, have been almost unanimous in the opinion that the choroid is derived from mesoderm, and this is the view taken by Kolmer [25] and Berens [26] (1936) in recent textbooks.

For the purposes of this paper I am especially concerned with the neurologic structure of the choroid. Agababow [27] was one of the earliest (1893) to investigate and describe the nerves of the sclera and choroid. He showed that the short ciliary nerves form a plexus in the posterior part of the eye, especially on the choroidal surface. True ganglion cells are seen here, the plexus gangliosus ciliaris of Krause. The long posterior ciliary nerves have only a minor part in the building of this plexus.

23. Geschickter, C. F., and Copeland, M. M.: Tumors of Bone, ed. 2, New York, American Journal of Cancer, 1936, p. 591.

24. Minot, C. S.: Human Embryology. New York, William Wood & Company, 1892, p. 710.

25. Kolmer, W., in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1936, vol. 3, p. 782.

26. Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936.

27. Agababow, A.: Ueber die Nerven der Sklera, Arch. f. mikr. Anat. **63**: 701, 1904; Ueber die Nerven in den Augenhäuten, Arch. f. Ophth. **83**:317, 1912.

Agababow further believed that the whole posterior half of the choroid to the exit of the venae vorticosae was traversed by ciliary nerves which sent fine branches to the suprachoroidal plexus and in which true ganglion cells were noted. He observed motor and vasomotor nerves in the choroid. The sensory nerve endings were in the sclera; none were seen in the choroid; however, he observed something analogous in the nerve network of the lamina suprachoroidea. Bietti's [28] investigations (1897) pointed to the conclusion that the nerves of the choroid have principally a vasomotor function.

Many later writers denied the occurrence of sensory fibers in the choroid.

In Salzmann's [29] "Anatomy and Histology of the Human Eyeball" (1912) it was stated that numerous nerve fibers, the last branches of the ganglionated plexus beginning in the suprachoroidea, traverse the choroidal stroma. In "The American Encyclopedia and Dictionary of Ophthalmology" [30] it was stated that many small branches derived from the ciliary nerves form a plexus in the lamina vascularis on their way through the suprachoroidea. They are provided with numerous large and multipolar ganglion cells showing origin from the sympathetic nervous system. Axenfeld [31] observed that the ciliary nerves pass through the sclera from the choroid in the anterior part of the globe. Nitsch [11] of the Vienna Eye Clinic said that sympathetic nerves and cell plexuses are normally present in the outside layer of the choroid. Berens [26] (1936) expressed the view that the long posterior ciliary nerves convey sympathetic fibers as well as sensory fibers. Berger and Vaillancourt [18] stated that Testut (1905) described and figured ganglion cells in the normal choroid and that they themselves observed such cells in the histologic investigation of the choroidal tumor in the case observed by them and were satisfied that they were of sympathetic origin. They expressed the opinion that the formation of sensory corpuscles is not confined to the central nervous system. All their investigations tended to show these writers that the ganglion cells form a peripheric neuron which is in connection with fibers coming from the ciliary nerves and that the choroidal tumor investigated by them originated from these ganglion cells.

28. Bietti, A.: Sulla distribuzione e terminazione delle fibre nervose nel corpo ciliare, Ann. di ottal. **26**:215, 1897; Anatomische Untersuchungen über die Regeneration der Ciliarnerven nach der Neurotonia optico-ciliaris beim Menschen, Arch. f. Ophth. **49**:190, 1900.

29. Salzmann, M.: Anatomy and Histology of the Human Eyeball, translated by E. V. P. Brown, Chicago, University of Chicago Press, 1912, p. 56.

30. Wood, Casey, A.: The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1914, vol. 3, p. 2161.

31. Axenfeld, T.: Nachweis und Bedeutung meiner "intraskleralen Ziliarnervenschleifen" am lebenden Merschenauge, Klin. Monatsbl. f. Augenh. **75**:602, 1925.

In connection with this question of sensory nerve fibers in the choroid, an interestnig observation has been made by Sédan.[32] This writer stated that in three patients with choroidal tumor whom he observed there was partial and limited anesthesia of the cornea in its external half, corresponding to the site of the lesion. The explanation, he thought, may be the fact that the tumor constricts the nerves in the suprachoroidal space by pressing them against the sclera and thus sensation is interrupted.

I have dwelt on the neurologic structure of the choroid because in connection with my own special work it is necessary to show that this vascular membrane is traversed by nerve elements, particularly the ciliary nerves, and that a neural neoplasm can easily arise in it, that is to say, that an ectodermal tumor can originate and proliferate in the midst of mesodermal tissue. As a matter of fact, a neoplasm containing neural elements is frequently observed in the uveal tract. Accordingly, I shall next deal especially with such neoplasms occurring in the choroid.

NEURAL TUMORS OF THE CHOROID

The most commonly reported tumors in the choroid are melanoma and so-called sarcoma. From what has been said earlier in this paper, melanoma, or melanotic sarcoma, may now be looked on as a neural tumor. Friedenwald [13] said that this tumor occurs in the choroid ten times as frequently as in the iris or the ciliary body. Miescher's [16] histologic investigations satisfied him that melanoma of the choroid originates in the choroid and not in the retina. Benign pigmented tumor of the choroid is rare, and generally the tumor becomes highly malignant.

Sarcoma of the choroid is perhaps the most important pathologic condition of the uveal tract. Annen [33] has classed it as pure melanosarcoma, pure leukosarcoma and mixed forms. Callender,[34] in an effort to arrive at a conclusion regarding its malignancy, classified so-called sarcoma according to cellular structure and cellular arrangement. According to "The American Encyclopedia and Dictionary of Ophthalmology," [35] sarcoma of the choroid is the chief intra-ocular growth of adult life, and its relative frequency in various clinics is from 0.03 to 0.06 per cent of that of all pathologic conditions of the eye.

32. Sédan, J.: Anesthésies cornéennes dans les tumeurs de la choroïde, Bull. Soc. d'opht. de Paris, December 1934, p. 620; Arch. d'opht. 52:595, 1935; Rev. gen. d'opht. 55:167, 1936.

33. Annen, E.: Sarcomes choroïdienes, Ann. d'ocul. 170:651, 1933.

34. Callender, G. R.: Malignant Melanotic Tumor of the Eye, Tr. Am. Acad. Ophth. 36:131, 1931.

35. Wood, Casey A.: The American Encyclopedia and Dictionary of Ophthalmology, 1914, vol. 3, p. 2173.

The literature shows that the origin of sarcoma of the bulb and its pathologico-anatomic nature are only partly understood, though, as Nitsch,[11] Schubert[36] and other writers suggested, this growth is probably due to malignant degeneration of neurofibroma. Nitsch remarked that sarcoma of the choroid always originates in the outside portion of the choroid and gives as the reason the fact that the nerves, for the most part, lie there.

In 1877 Knies[37] made a report of the histologic study in sixteen cases of sarcoma of the choroid. In all these cases he observed that the tumor had its origin in the posterior section of the bulb and, more precisely, in the middle layers of the choroid. He cited Wecker as asserting that melanosarcoma most frequently arises in the anterior portion of the bulb, anterior to the equator. Wintersteiner[38] in 1900 reported a case of sarcoma of the choroid originating in Haller's layer, that is, the layer of large vessels. Schubert[36] in 1925 reported a case of leukosarcoma which he thought could be classified as an instance of tumor due to malignant degeneration of neurofibroma. I cite this case because the tumor seemed to Schubert to be a neurogenous growth the origin of which was in the long posterior ciliary nerve.

Reference to the possibility of sarcoma arising from a nevus was made in Parsons'[12] "Pathology of the Eye" (1905). Schieck's[39] pathologic and histologic description in 1906 of a sarcomatous degenerating nevus of the right iris is interesting, as it shows that at this early date pathologists recognized that sarcoma can develop from a nevus. This was, of course, prior to Masson's[9a] observation that nevi have a neural origin. In 1928 Wätzold and Gyotoku[40] published a histologic study of twenty-seven cases of sarcoma of the choroid, in only five of which the tumor originated in the ciliary body and extended into the choroid. The significant point is this: These investigators considered that choroidal sarcoma, owing to the alveolar structure and polymorphous cellular forms, suggests an ectodermal origin which, however, they concluded can be successfully established only if cellular elements of like derivation are proved to be present in the choroid. It is now known that such neural elements do occur in the choroid. Another inter-

36. Schubert, F.: Operation eines "Leukosarcoms" der Choroidea mit Erhaltung des Auges, Ztschr. f. Augenh. **56**:252, 1925.

37. Knies, M.: Sixteen Cases of Sarcoma of the Choroid, Arch. Ophth. & Otol. **6**:376, 1877.

38. Wintersteiner, H.: Beobachtungen und Untersuchungen über den Naevus und das Sarkom der Conjunctiva, Ber. u. d. Versamml. d. ophth. Gesellsch. **27**:253, 1899.

39. Schieck, F.: Das Melanosarkom als einzige Sarkomform des Uvealtraktus, Wiesbaden, J. F. Bergmann, 1906, case 24.

40.. Wätzold, P., and Gyotoku, K.: Zur Pathogenese des Aderhautsarkoms, Arch. f. Ophth. **120**:209, 1928.

esting item in this report is the fact that only one of the twenty-seven growths was a leukosarcoma, the other twenty-six being pigmented tumors.

Cases of pure neuroma, sometimes termed neurofibromatosis, pigmented or unpigmented, of the choroid, have been reported. Snell and Collins [41] (1903) are credited as being the first to show that the choroid can be the site of tumors in neurofibromatosis of the ciliary nerves, and they described a plexiform neuroma in which the ciliary nerves were thickened by what at that time they considered as perineural and endoneural proliferation of connective tissue; the thickening of the choroid was also considered as showing that the ciliary nerves could be attacked by neurofibromatosis. Other cases of ocular neurofibromatosis have since been reported, by Sutherland and Mayou [42] in 1907, Murakami [43] in 1913, and Kyrieleis [44] in 1928.

Fuchs [45] observed two neuromas of the ciliary nerves composed almost exclusively of medullated nerve fibers. In one case there was a knotlike thickening of one of the ciliary nerves lying in the choroid.

In 1925 Dr. Mary Knight,[46] of the Mayo Foundation, reported a melanotic tumor of the choroid; the article had excellent illustrations showing the histologic observations. The thickening of the choroid was due, in the author's opinion, to excess of connective tissue. She considered the case as one of fibroblastoma or meningoblastoma. Her investigations convinced her that the chromatophores of the choroid do not produce pigment but are merely carriers of it. This would accord with Miescher's [16] observations, already alluded to, except that Miescher believed that under neoplastic conditions the chromatophores can revert to their original embryonic function and again produce pigment.

Berger and Vaillancourt [18] in 1934 reported a case of tumor of the posterior segment of the choroid. The tumor originated intrachoroidally. After an elaborate histologic study the authors arrived at the conclusion that the tumor was a melanogenous ganglioneuroma with sensory corpuscles, and they considered it to correspond with Masson's [9a] neuronevus of the skin. From a study of their histologic descriptions and

41. Snell, S., and Collins, E. T.: Plexiform Neuroma of Temporal Region, Orbit, Eyeball and Eyelid, Tr. Ophth. Soc. U. Kingdom 23:157, 1903.

42. Sutherland, G., and Mayou, M.: Neurofibromatosis of the Fifth Nerve with Buphthalmus, Tr. Ophth. Soc. U. Kingdom 27:179, 1907.

43. Murakami, S.: Zur pathologischen Anatomie und Pathogenese des Buphthalmus bei Neurofibromatosis, Klin. Monatsbl. f. Augenh. 16:514, 1913.

44. Kyrieleis, W.: Ein Neurinom am Limbus corneae, Arch. f. Ophth. 119:119, 1928.

45. Fuchs, E.: Ueber schaumige Einlagerunge in der Aderhaut, Arch. f. Ophth. 118:697, 1927.

46. Knight, Mary S.: A Critical Survey of Neoplasms of the Choroid, Am. J. Ophth. 8:791, 1925.

illustrations Berger and Vaillancourt considered that the choroidal tumors reported by Snell and Collins [41] and by Knight [14] were counterparts of the tumor observed in their case; they found no reports in the literature of others of exactly the same kind.

With the exception of Berger and Vaillancourt's neuronevus of the choroid, none of the authors cited referred to or gave histologic evidence of the origin of choroidal neoplasms by proliferation of the Schwann sheath cells.

Dr. Algernon B. Reese loaned a section of an eye in which a nevus of the choroid and long posterior ciliary nerve was found on routine examination (fig. 1 *A* and *B*). The nevus was in no way responsible for the enucleation and on account of its location was not suspected.

The section was stained with hematoxylin and eosin. The eye had been cut in an almost true horizontal plane, the sections showing segments of the long posterior ciliary nerves on both the temporal and the nasal side. On one side the long posterior ciliary nerve was normal and as it neared the choroid was accompanied by only a few chromatophores. On the opposite side the nerve channel ran through the sclera in a long diagonal course. In its proximal part the channel contained normal-appearing nerve and part of a vascular wall accompanied by a few pigmented cells. The greater part of the canal was occupied by a nevus which widened the canal and ended in a nodule of heavily pigmented cells in the choroid, where these pigmented cells continued anteriorly for a short distance. Inside the scleral channel the cells, for the most part, were parallel with it, and as they reached the choroid they swirled about, assuming a position parallel to it. The nevus was composed of various cells: Long pigmented cells with faint nuclei strung along chiefly in the peripheral part of the canal but were seen throughout the nevus; there were long, blue-staining spindle cells with thin nuclei, and short, blunt spindle cells with large nuclei; the round pigmented cells varied in size from small to very large. The nodule in the choroid was so dense that the individual cells could not be recognized, but outlines of round, spindle and branched pigmented cells coulld be detected.

Occurring in the path of this large nerve—and recalling the observations of Masson,[9a] Foot [20] and others—this nevus, it can be said, undoubtedly arises from the Schwann sheath cells and the pigmented cells which normally may outline the ciliary nerves and vessels. It is possible that a malignant tumor may arise from just such a nevus of the ciliary nerve and choroid.

The structure and arrangement of the cells in uveal sarcoma have been described well in past literature on the eye. The derivation of the spindle cells in fascicular and palisade formations of the more anaplastic pleomorphous epithelioid type of cells and of the various types of pigment cells has been stated to be mesoderm.

So far as I know, the specimens which I am about to describe are the first in which histologic evidence is furnished of the neural origin of so-called sarcoma of the choroid, that is to say, its origin from proliferating Schwann sheath cells of the ciliary nerves traversing the choroid.

PERSONAL WORK ON SARCOMA OF THE CHOROID

Dr. Richard Jaffé, of Chicago, after reading the work of P. Masson [9] concerning the neural origin of certain types of sarcoma, suggested that I search for nerves in relation with sarcoma in the choroid, where this

Fig. 1.—*A*, nevus of long posterior ciliary nerve and choroid. *B*, high power photomicrograph of the nevus.

type of tumor is frequent. At that time (1932) I had sectioned serially and stained every section of a bulb specimen (7) in the horizontal meridian, for the study of its normal anterior segment.[47] To the

47. Dvorak-Theobald, G.: Schlemm's Canal: Its Anastomosis and Anatomic Relations, Tr. Am. Ophth. Soc. **32**:574, 1934.

temporal side of the optic nerve, in the choroid, a tumor was observed, composed mostly of nonpigmented spindle cells; pigmented cells were seen in the periphery of the tumor and were sparsely present between bundles of cells. In this tumor, through sixty sections—almost 1 mm.— the long posterior ciliary nerve was seen intimately involved in the growth. It entered the tumor with well defined boundaries; it seemed to burst and bloom by proliferation of its Schwann sheath cells to form the surrounding tumor, and the nerve then continued forward on its accustomed way. The tumor was composed of spindle cells in bundle and sheet formation and was classified as a leukosarcoma.

Since that time a number of other bulbs enucleated because of a choroidal tumor have been sent to the laboratory.

The most frequent site of a uveal tumor is to either side of the optic foramen in the horizontal meridian, that is to say, in the path of the long posterior ciliary nerves; when it is farther forward it is off the horizontal plane, in the upper or lower nasal or temporal quadrant. Trying to trace nerves into a more anterior tumor would be a tedious task, as it might arise from any of the numerous short ciliary nerves or branches of the long posterior ciliary nerves. In all, seven bulbs were selected, sectioned serially and stained with hematoxylin and eosin, Masson's trichrome stain, Mallory's stain for connective tissue, Van Gieson's stain, Held's stain and Foot's silver stain. Six bulbs were cut as horizontally as possible so as to obtain longitudinal sections of the long posterior ciliary nerves. In five of these bulbs the Schwann sheath cells of the long posterior ciliary nerves were observed to be proliferating to form the tumors. The sixth bulb showed no involvement of the long posterior ciliary nerve. A small tortuous ciliary nerve traversing the choroid between the optic nerve and the long ciliary nerve was involved in a nevus formation. Because several important sections were lost in sectioning this bulb, the exact relation of this nevus to the tumor cannot be definitely known.

The seventh bulb was cut diagonally because of a sarcoma in the posterior upper temporal quadrant. By accident sections were cut longitudinally through a small ciliary nerve which ran through fifteen sections. As this nerve neared the choroid its Schwann sheath cells became more numerous and stained more brilliantly than the cells of the normal nerve. At its very entrance into the choroid the Schwann sheath cells proliferated into the tumor. All these sections showed the axons to be few and scattered. Other ciliary nerves passed through the tumor without change.

According to the present day nomenclature, the tumors here described are classified as spindle cell sarcoma and leukosarcoma; melanosarcoma, and mixed cell sarcoma. It was observed that nonpigmented tumors had their origin from Schwann sheath cells of nerves

which were accompanied in the scleral channel by few or no pigmented cells and that the pigmented tumors originated from the pigmented cells surrounding the nerve, the peripheral Schwann sheath cells or those of the entire nerve.   In the cases in which the last origin was noted the normal long posterior ciliary nerve on the opposite side was accompanied by pigmented cells.

These tumors were so-called sarcomas of the choroid of various cellular structure—spindle cell, round cell, melanotic cell and mixed cell—in varying stages of maturity, some having chiefly mature spindle cells and others pleomorphous anaplastic cells.   In some cases the tumor began in the nerve as it passed through its scleral channel; in others the tumor originated from the nerve intrachoroidally.

The microscopic observations in the aforementioned cases are given in the following section.   As fine descriptions of the structure and cytologic features of similar tumors are to be found in the literature, I

Data for Seven Cases of So-Called Sarcoma of the Choroid

Date	Surgeon	Speci-men	Name of Patient	Sex	Age, Years	Duration of Symptoms Prior to Operation	Pathologic Diagnosis
1932	Dr. George Suker	1	......	F	52	5 mos.	Spindle cell sarcoma
1/13/36	Dr. Harry Woodruff	2	F. E.	F	58	2 yrs.	Spindle cell sarcoma
1/17/35	Dr. T. D. Allen	3	D. S.	M	52	2 mos.	Melanosarcoma
7/19/35	Dr. Theodore Shapira	4	B. S.	M	32	?	Melanosarcoma
5/23/35	Dr. E. K. Findlay	5	R. M.	M	61	2 yrs.	Mixed cell sarcoma
7/12/35	Dr. T. D. Allen	6	E. J.	M	..	2 yrs.	Spindle cell sarcoma
7/—/—	Dr. R. J. Curdy	7	K. S.	M	..	....	Spindle cell sarcoma

shall limit my own descriptions as much as possible to the relation of the nerves to the tumors.   As the patient's history is not pertinent to the observations here described, only enough is given to identify the specimen; also description of the gross and microscopic appearance of the entire eyeball is omitted.

MICROSCOPIC OBSERVATIONS

SPECIMEN 1.—The pathologic condition in this eye depended entirely on the choroidal tumor, which had broken through the lamina basalis and was infiltrating the retina. The tumor was composed mostly of nonpigmented spindle cells; spindle cells were noted in its periphery and were sparsely present between bundles of cells. The tumor was slightly vascular, becoming more vascular after breaking into the retina, where the vessels were almost cavernous. Sections 340 to 400 showed interesting details concerning the relation of the long posterior ciliary nerve to the tumor.

As was mentioned previously, the sections were cut parallel with this nerve, and it could be traced through these sections from its entrance to its exit and beyond (fig. 2 A). As the nerve entered the tumor its boundaries were well defined for a short distance. It widened; its boundaries broke, and the Schwann sheath cells proliferated, giving rise to the surrounding tumor (fig. 2 B). The cells nearest the ciliary nerve were very mature, long spindle cells with pointed nuclei.

A

B
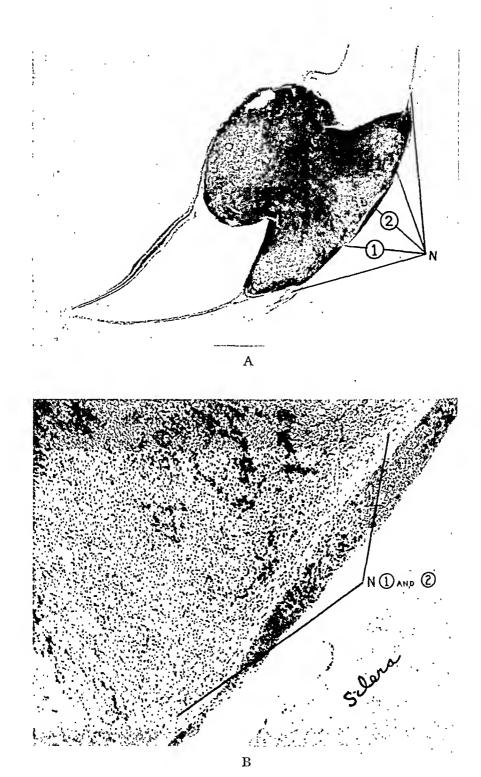
Fig. 2 (Specimen 1).—In *A*, *N* marks the course of the long posterior ciliary nerve. At *1* and *2* the Schwann sheath cells are proliferating to form the tumor. *B* shows the proliferation of Schwann sheath cells into sheets and bundles of tumor cells, growing almost at right angles to the nerve.

The cells grew into intertwining sheets and bundles, which were almost at right angles to the course of the nerve. The newer cells were shorter. The oval nuclei had one or more nucleoli. Where the tumor had broken through the lamina basalis the cells were very anaplastic and mitosis was active. Pigment cells were seen in the periphery of the tumor and between the bundles of cells.

With Masson's trichrome stain the Schwann sheath cells and the tumor cells were colored red, as were all other known ectodermal tissues. The cornea and sclera and the connective tissue about and lining the capillaries and various septums stained blue. The collagen, Masson said, is not mesodermal, but is determined by the Schwann cells, just as basement membrane is determined by epithelium. Sections stained with other methods corroborated this differentiation of the tissues. No masses of new connective tissue or granulation tissue were observed.

Summary of Histologic Study.—The growth was a malignant, only slightly pigmented tumor, originating intrachoroidally from the Schwann sheath cells of a long posterior ciliary nerve. The tumor was what has been commonly described as spindle cell leukosarcoma.

SPECIMEN 2.—This tumor was composed of large spindle cells with well defined nuclei and nucleoli. The cells grew in sheets and bundles which had only a small amount of collagenous material between them. Cells grew in whorls about the vessels, most of which were lined by collagen with endothelium. This was well brought out by the Foot silver stain. Pigmented cells in small numbers were seen near the sclera; they were mixed cells—spindle, round and chromatophore-like— of varying sizes and contained both fine and coarse granules of pigment.

Sections 290 and 381 passed through the long posterior ciliary nerve. As the nerve entered the scleral channel it was accompanied by a few pigmented cells (fig. 3 *A*). Histologically, it appeared normal, with the axons, Schwann sheath cells and collagenous fibers in perfect order. After it wound over a larger artery its character changed; the nerve appeared in disorder, and the cells varied— there were long spindle cells with or without pigment; thick, blunt spindle cells with indefinite nuclei, like those found in nevi, and cells with well defined nuclei and nucleoli, like those found in the main body of the tumor. Winding through these cells were strands of normal Schwann sheath cells and capillaries. The axons, which are well seen in the normal nerve, were absent (shown by Held's stain). As the Schwann sheath cells progressed into the choroid, tumor cells with shorter and more blunt nuclei grew from them (fig. 3 *B*). These cells in turn gave rise to others which were oval or even round.

With Masson's trichrome stain the normal Schwann sheath cells and the tumor cells took a red color; the axons stained red, and collagenous fibers stained blue. All the known ectodermal tissues stained red, and all the known mesodermal tissues stained blue. With Foot's silver stain only the collagenous fibers between bundles of cells and the outlines of vessels appeared pinkish brown; the tumor cells and the Schwann sheath cells did not stain. As serial sections of this tumor were studied the impression of bundles of Schwann sheath cells twining and intertwining became most apparent. As the process continued the cells became more anaplastic.

Summary of Histologic Studies.—This growth was a malignant, only slightly pigmented tumor originating from the Schwann sheath cells of a long posterior ciliary nerve and its accompanying pigment cells; the nerve was tumorous while still in the scleral channel. The tumor was what has been commonly described as spindle cell sarcoma.

A

B
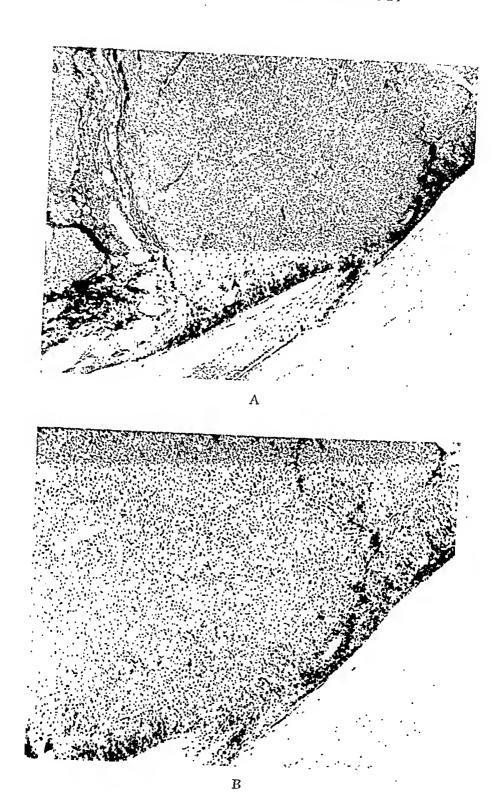
Fig. 3 (specimen 2).—A, tumorous nerve traversing the scleral canal. B, higher magnification of A, showing pigmented cells in the nerve while still in the scleral canal.

SPECIMEN 3.—The tumor was composed of mixed cells. The largest part was composed of pigmented spindle cells of varying sizes; here the vessels were large and almost cavernous; some of these vessels were lined with endothelium, others with collagen. The smaller part of the tumor was composed of unpigmented cells and very slightly pigmented cells, more closely packed together in sheets and bundles.

The long posterior ciliary nerve could be traced through sections 121 to 200. When the nerve entered the scleral channel it was accompanied by pigmented cells, which increased in number, size and content of pigment as the nerve neared the choroid (fig. 4). At this point the peripheral Schwann sheath cells began to proliferate into spindle cells which had more or less pigment. This could best be seen where the section had been slightly pulled in cutting and mounting. Large round cells with heavy pigment accompanied them; many of these cells had processes or feet that hugged the nerve. Farther on the most peripheral Schwann sheath cells and pigment cells proliferated to several layers; ·the unpigmented cells became blunt and rounded, and the chromatophores became round and had coarse granules, and their nuclei had one or more nucleoli. Division of cells was by amitosis. The nerve became thinner as it proceeded through the tumor and disappeared.

Comment.—In this specimen, in contrast to the two previously described, the perineural pigment cells, as well as the Schwann sheath cells, proliferated to form the tumor. These pigment cells, Miescher [16] showed, are also derived from neural ectoderm.

Summary of Histologic Study.—This growth was a malignant tumor of the choroid, originating in the Schwann sheath cells of a posterior ciliary nerve and its accompanying pigment cells. The tumor originated in the scleral channel. It was of the kind commonly described as melanosarcoma or mixed cell melanosarcoma.

SPECIMEN. 4.—The tumor was necrotic, and massive hemorrhages had occurred, probably after an operation for the relief of tension. Mixed cells comprised this tumor. Thin spindle cells became more blunt and wide where growth was rapid. There were large oval or round unpigmented cells with from one to three nucleoli in the large nucleus, which may be called epithelioid cells. Some of the spindle cells were pigmented. There were round pigmented cells of varying sizes with fine and coarse granules. The vessels were lined for the most part by collagenous fibers. There was a large extrabulbar extension between the sclera and Tenon's capsule, in which the nest of epithelioid cells were separated by wide collagenous bands.

The long posterior ciliary nerve was studied in relation to the tumor in sections 260 to 360. This nerve was of interest while still in its scleral channel (fig. 5 A). It was wide, and there was a marked decrease in the axons. The Schwann sheath cells were increased in number and irregular in arrangement. The external layers of these cells were proliferating and mingling with pigment cells which were massed about the nerve. As the nerve progressed into the tumor it became thinner, the Schwann sheath cells were more regular, and occasional segments of axons were seen. From the inner surface of the nerve the Schwann sheath cells and pigmented cells proliferated into sheets and bundles (fig. 5 B). These cells were blunt spindle cells which became more mature or anaplastic as the tumor progressed away from the nerve. With Masson's trichrome stain the Schwann sheath cells, as well as the tumor cells, stained red— the protoplasm a light red, the cellular and nuclear walls and the nucleoli a dark red. Foot's

A

B
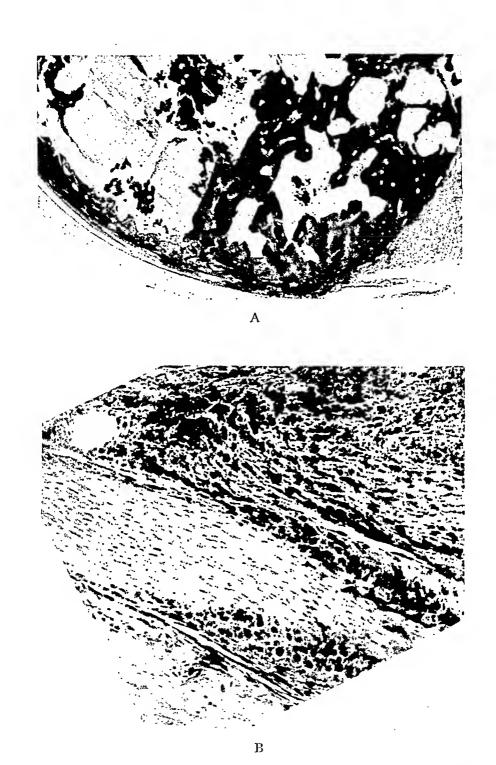
Fig. 4 (Specimen 3).—*A*, long posterior ciliary nerve entering tumor. *B*, detail of *A*, showing proliferation of tumor cells from the Schwann sheath cells and accompanying pigment cells.

A

B
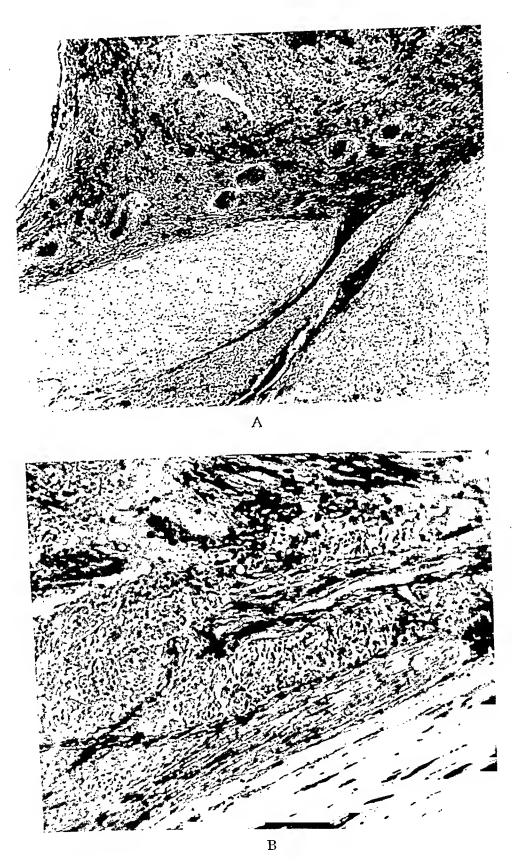
Fig. 5 (Specimen 4).—In *A* the long posterior nerve is tumorous while still in the scleral canal. *B*, tumorous nerve showing pigment cells and proliferation of its peripheral cells into tumor cells.

silver stain silhouetted the collagenous fibers seen between cells and bundles of cells and lining the vessels. Held's stain was best for the axons. The long posterior ciliary nerve of the opposite side was normal and was accompanied through the scleral channel by pigmented cells.

Summary of Histologic Study.—This growth was a malignant tumor of the choroid, originating in the Schwann sheath cells of a long posterior ciliary nerve and its accompanying pigment cells while it was still in the scleral channel. It was of the type commonly diagnosed as melanosarcoma.

SPECIMEN 5.—The choroid on each side was occupied by a tumor; in the sections studied these tumors had no connection with each other. There were metastatic growths in the vitreous and the sclera, so it was thought that perhaps a connection would be found in other sections or in the calottes. The greater part of the choroidal tumors was necrotic. The live cells were in the periphery of the tumor; they were large, polymorphous cells with large nuclei and one or more nucleoli. They divided by both mitosis and amitosis. From their outlines, the necrotic cells were judged to be more spindle shaped. On the temporal side the long posterior ciliary nerve was converted into tumor, only a few central normal Schwann sheath cells being recognizable (fig. 6 A). On the nasal side the nerve passed through the tumor with its boundaries well intact and remained independent of the tumor (fig. 6 B). The staining properties of the cells of this tumor were identical with those of the tumors already described.

Summary of Histologic Study.—This growth was a malignant tumor of the choroid, originating in the Schwann sheath cells of a long posterior ciliary nerve and its accompanying pigment cells. It was of the type commonly diagnosed as mixed cell sarcoma.

SPECIMEN 6.—The tumor was a typical spindle cell sarcoma with fascicular cellular arrangement. The staining properties were identical with those of the previously described tumors. This eye was not cut truly horizontally, and tracing the nerves was unsatisfactory and difficult. The long posterior ciliary nerve, which was traced through sections 260 to 340, was found undisturbed by the surrounding tumor (fig. 7 A). While still in the scleral channel its peripheral Schwann sheath cells proliferated into several layers (fig. 7 B). Smaller nerves entered the tumor, accompanied by vessels, but their relation to the tumor could not be determined. A small tortuous ciliary nerve and its vessel, traversing the sclera between the optic nerve and the long posterior ciliary nerve, were involved in a nevus formation. The choroidal tumor may have had its origin in this nevus, but because several important sections were lost in cutting it cannot be definitely so stated.

Comment.—I am including this case to show that it is difficult at times to trace the nerves into the tumor. The nerve must be cut longitudinally and not on the bias. It is also entirely possible that many normal nerves course through the tumor, while the tumor originates from one.

Summary of Histologic Study.—This growth was a malignant tumor of the choroid, originating probably from the nevus in the sclera. This was a so-called spindle cell sarcoma.

SPECIMEN 7.—As was stated previously, this bulb was cut diagonally because the tumor was in the upper temporal quadrant in the posterior segment. The tumor was composed of spindle cells in fascicular arrangement. The cells nearest the sclera were long, slender spindles with dark nuclei, in which details could not be seen; these cells grew in sheets and bundles. As the tumor grew the cells

A

B
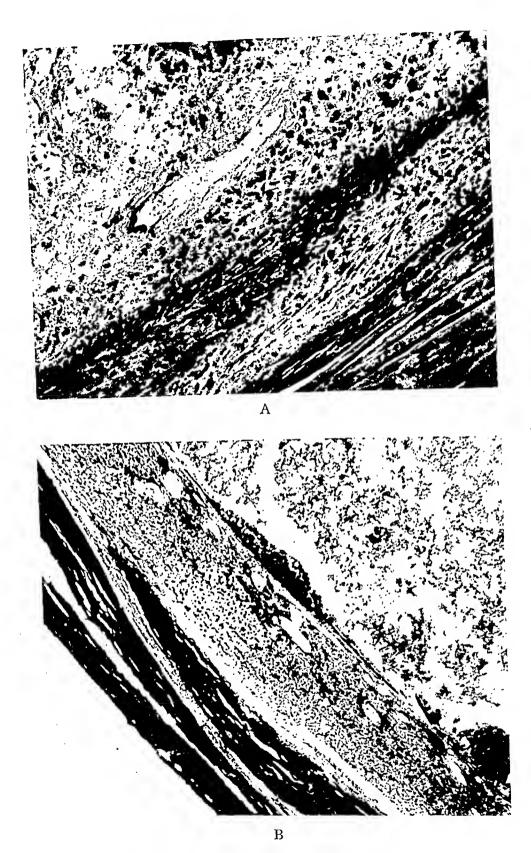
Fig. 6 (Specimen 5).— In *A* the long posterior ciliary nerve is shown converted into tumor, only a few central normal Schwann sheath cells being recognizable. In *B*, the nerve passes through the tumor with its boundaries well intact and remains independent of the tumor.

A

B
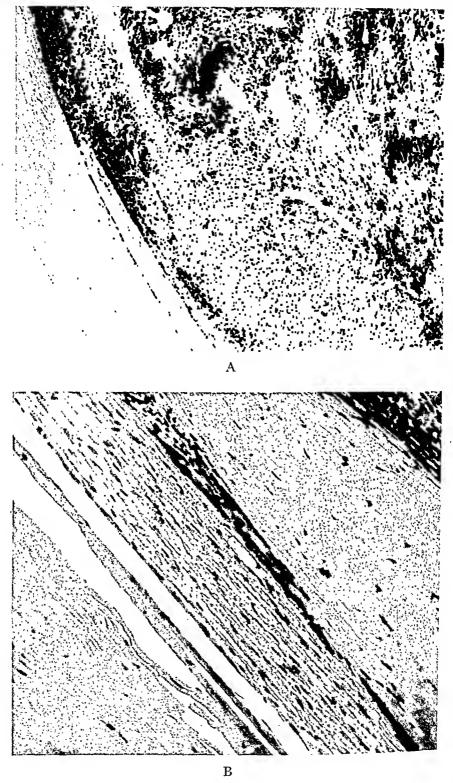
Fig. 7 (Specimen 6).— *A* shows the long posterior ciliary nerve traced through sections 260 and 340, undisturbed by the tumor. *B* shows that while still in the scleral channel the peripheral Schwann sheath cells of this nerve proliferated into tumor cells.

became shorter and the nuclei more blunt, and one or more nucleoli were seen. With further growth the cells became almost round and pleomorphous but were still in fascicular and palisade formations about vessels and spaces lined with collagen. Pigment cells were few, were present in the periphery of the tumor and had no part in its formation. The outside mature cells grew in sheets parallel to the surface of the tumor, forming a capsule for the fasciculated bundles of more anaplastic cells. This sheath broke anteriorly, and the new cells grew through the lamina basalis and invaded the retina. Sections 149 to 170 showed a short ciliary nerve coursing through the sclera in a long oblique path and entering the tumor (fig. 8 *A*). The nerve was accompanied by only a few pigment cells. Its Schwann sheath cells were normal in appearance and in number until the nerve neared the choroid. Here the Schwann sheath cells increased markedly
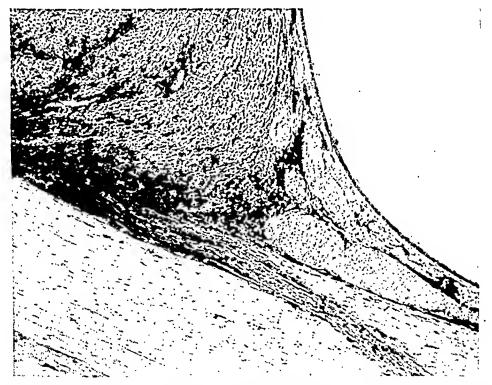
Fig. 8 (Specimen 7).—A short posterior ciliary nerve becomes tumorous as it nears the choroid, and gives rise to the tumor in it.

in number; they were long, thin spindle cells with heavily pigmented nuclei and continued on, blending with the tumor (fig. 8 *B*).

Summary of Histologic Study.—This growth was a malignant tumor of the choroid, originating in the Schwann sheath cells of a short ciliary nerve. It was a so-called spindle-cell sarcoma.

GENERAL COMMENT

Small choroidal sarcomas are disklike, and their peripheral cells form a laminated capsule. As the tumor grows the inner surface breaks and the tumor cells, being unconfined, grow rapidly into the characteristic mushroom shape. In the early stage sarcoma is much like the encapsulated peripheral nerve tumor the capsule of which is formed of

lamellae. Early authors recognized the fascicular type, which may show palisades, and a reticular type, and realized that despite these aspects the neurinomatous tissue is a unity. Masson [9b] showed that the palisaded formations in neurinoma are not accidental but are organoid products of sensory nerves, while nonpalisaded neurinomas are products possibly of motor nerves. The pathologic picture of various so-called choroidal sarcomas differs because of the fact that ciliary nerves carry both sensory fibers and motor fibers.

One can see by the study of these specimens that the syndrome of Sédan must occur not from pressure of the tumor on the nerve but by the interruption of the axons and the proliferation of the schwannian elements.

My observations that choroidal tumors originate from the Schwann sheath cell and the accompanying pigment cells is in general accord with the observations of Masson [9a] and of Berger and Vaillancourt, who identified pigment cells with Schwann cells. The finding corroborates that of Berger and Vaillancourt,[18] who said that it is an absolutely incontestable fact that in neoplastic conditions the nerve cells or the syncytium of the Schwann sheath cells can assume the property of melanogenesis. Nevertheless, the process of differentiation of pigmented neoplasms from unpigmented neoplasms under the circumstances would seem to call for further investigation.

I submit that I have proved the main point at issue, that is, the neural origin of so-called sarcoma of the choroid. The name sarcoma applied to neural tumors of the malignant type is a misnomer; it connotes connective tissue, and certainly these neural neoplasms are based on neither connective tissue nor epithelial tissue. Sarcoma was also distinguished by the spindle cell structure of tumors, but one sees that spindle cells may occur in tumors arising from neural ectodermal tissue as well as in those arising from mesodermal tissue. It is time to revise the classification of neoplasms, distinguishing them on the basis of embryonic tissue rather than on the theoretical basis of structure and cellular characteristics. As Broders and MacCarthy [15] justly observed: "Morphology is not an accurate criterion for embryologic origin in any specific embryonic layer. Moreover, the least differentiated tissues, such as the embryonic tissues, are more related to the tissues of tumors than to the tissues of normal organs."

SUMMARY AND CONCLUSIONS

The nature of neurogenic neoplasms in general is discussed, with particular reference to Masson's histologic investigations regarding cutaneous nevi and tumors of the peripheric nerves.

The literature regarding melanotic neoplasms and the origin of pigment is reviewed.

The neurologic structure of the choroid and neural tumors of the choroid, including those termed sarcoma, is described.

Original observations by myself in seven cases of so-called sarcoma of the choroid are given in detail, and from the histologic observations it is submitted that these tumors originated from the Schwann sheath cells of the posterior ciliary nerves in their passage through the choroid.

A revised classification of neoplasms is suggested, which would distinguish ectodermal neural tumors from those of other embryonal origin.

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Clinical Notes

DUGAS ON THE REMOVAL OF FOREIGN BODIES FROM THE EYE

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Dr. Louis Alexander Dugas, M.D., LL.D. (1806-1884), held the chair of surgery at the Medical College of Augusta (the present University of Georgia School of Medicine) from 1855 to 1882, was dean for a period of twenty years and editor of the *Southern Medical and Surgical Journal* from 1851 to 1858. Eponymously he is remembered for his famous sign indicative of dislocation of the shoulder joint. During his professional career he published approximately one hundred articles which are a credit to his surgical versatility and the wide range of his medical knowledge. The paper which follows is one of several Dugas manuscripts which have recently come to light.

A SIMPLE PROCESS BY WHICH MOTES AND OTHER FOREIGN BODIES MAY BE REMOVED FROM THE EYE

By Louis A. Dugas

Read before the Augusta Library and Medical Society
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The removal of motes or fragments of foreign bodies from the external Surface of the eye is an operation we are frequently called upon to perform.

Rail Road employes and travellers, workers in metals and Stone Cutters, are those most frequently claiming our Services. The natural Sensitiveness of the external eye is usually very much increased when we are consulted. So that the intolerance of light makes it difficult to examine the eye thoroughly.

With a little careful manipulation however we may succeed in finding the foreign body upon the cornea, the ocular conjunctiva or beneath the eyelids. Whenever found it is more or less difficult to remove by the procedures usually recommended by written authorities, and which you know consists simply in its removal by means of one or other form of instrument while the eyelids are held open. No directions are given for the purpose of rendering the eye motionless during the operation. And yet it is extremely difficult for the surgeon as well as painful to the patient to dislodge the foreign body while the eye is instinctively avoiding every approach of the instrument. In order to Surmount this difficulty I have for many years been in the habit of placing the end of the index finger upon the eye just within the canthas, and retaining it there until I have removed the object. The contact of the finger produces a sensation which, while not decidedly painful, is yet sufficiently decided to engross the attention of the patient and to prevent his moving the eye at the approach of the instrument or on its contact with the ocular surface.

By this plan the foreign body may be removed from the surface of the eye as readily as from any other part and without the risk of Scratching or otherwise injuring the organ by repeated and unsuccessful attempts to take it by surprise, if I may use the expression, by sudden thrusts of the instrument used for the purpose. I am in the habit of using Scarpa's cataract needle, and find it better adapted to the purpose than any other instrument, whether the mote be embedded or in Simple contact.

A young man accompanied by his father came from one of our upper counties to get me to remove a thorn sticking in his cornea. It seems that he was walking in the garden and passing by a rose vine when a branch coming in contact with his eye one of the thorn plunged into the cornea and was left there by the onward movement of the young man. He applied to the physicians of the neighborhood, one after another who made unsuccessful attempts to remove it with pocket knives, bistouris, lancets, etc. When he arrived here I found him badly scarred and very despondent. By putting the end of my fingers upon the eye-ball so as to keep it quiet the thorn was at once removed without any difficulty whatever—I may add that the delighted father exclaimed: "How strange it is that no one else thought of this Simple method of proceeding."

I am not prepared to say that no else has ever resorted to this method, but I have been teaching its advantages very many years to the classes of the Medical College of Georgia.

It is more than probable, that other surgeons have used it, and, like myself, omitted to publish their views.

Ophthalmologic Review

SURGICAL TREATMENT OF STRABISMUS

REVIEW OF THE RECENT LITERATURE

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NEW YORK

One hesitates to add to the great bulk of literature on the surgical treatment of strabismus. Yet it is the vast and ever increasing number of articles on this subject which has made it seem desirable to attempt a summary of the more recent reports so that the busy surgeon may have some idea of the present status of this important branch of ocular therapy. Practical considerations have made it necessary to limit this review to the literature of the last seven years (from 1930 through 1936). A conscientious attempt has been made to include everything written on the subject during this period, to point out the important contributions and to give the opinion of the majority wherever possible.

For the purpose of this review it has seemed expedient to divide the material into two groups: that on nonparalytic strabismus and that on paralytic strabismus. In the section on the surgical treatment of nonparalytic strabismus the various operations for weakening and strengthening the action of the ocular muscles are discussed. In the section on paralytic strabismus the operation of choice for each muscle is given.

NONPARALYTIC STRABISMUS

Complete Tenotomy.—This procedure has few exponents today. Carroll and Blake in their splendid research on rabbits pointed out that the results of tenotomy are unpredictable because of the failure of the muscle to become reattached to the globe in some instances. Peter considers complete tenotomy dangerous. In his detailed analysis Folk concluded that complete tenotomy in convergent strabismus is uncertain and leads to divergence, while in treating divergence there is no danger. The great uncertainty of the results and the occasional severe overcorrection have caused the majority of writers to abandon tenotomy in favor of one of the more controlled operations. However, some writers still advocate it. Hepburn has protested against the wholesale condemnation of tenotomy. He considers this operation very useful.

especially for young children who require a general anesthesia. Bufill believes that simple tenotomy has often given excellent results. Cosgrove, Argañaraz and Perwög favor bilateral tenotomy of the internal rectus muscles in cases of severe strabismus. Onfray uses tenotomy only when there is no hope of binocular vision without it and then waits until the patient is 16 or 18 years of age before operating. Terrien, while admitting that this operation has been too extensively employed, stated the opinion that it should be retained, with the following reservations: It should be used only for strabismus of very high degrees; it should be limited to one muscle; the lateral attachments should be preserved; it should never be used before the patient is 10 or 12 years of age, and it should be used with an advancement, the aim being toward overcorrection.

Partial Tenotomy.—Opinions as to the value of partial tenotomy vary widely. Some writers (Peter, Folk) consider it practically useless, while others have advocated its use, usually in conjunction with one of the shortening operations (Burch and Grant, Castresana, Lordan, Hosford and Hicks, O'Connor). Stevens' central tenotomy is still being used by Hosford and Hicks and by Lordan. O'Connor described a two stage tenotomy for use with his cinch shortening operation. The first stage is central tenotomy. Six weeks later, after the central portion of the muscle has become reattached, he cuts the margins. He placed emphasis on maintaining control and avoiding torsion. Pollock has given the only description of tendon lengthening, performed by a modification of the method of Bishop Harman.

Various methods are in use for controlling the effect of tenotomy. While the details vary widely, the fundamental principle is to place a suture in the muscle or tendon before performing the tenotomy and then to secure it more or less firmly to either the stump (Thaw, Gorst) or the overlying conjunctiva (Alexiadès, Goldenburg). Usually the suture is tied lightly and left long for later adjustment. Folk considers guarded tenotomy less safe than partial tenotomy because of the danger of sutures slipping.

Recession.—The various types of tenotomies have largely been replaced by the recession (retroplacement) operation in recent years. Judging from the reports in the literature, the recession operation is the most popular operation on the ocular muscles today, used either alone or in conjunction with a shortening operation. The fundamental principle of this procedure is firm anchorage of the tenotomized tendon to the sclera at a measured distance behind the original insertion. It is to the splendid, painstaking work of Jameson that ophthalmologists are largely indebted for this principle and for the carefully detailed technic that has led to its wide acceptance. The critics of this operation

point out the danger of perforating the sclera with the needles, with resultant intra-ocular infection, and setting the muscle too far back, which causes crippling of convergence. That both mishaps can occur is undenied, but that they should not occur if the proper technic is followed is the claim of the many supporters of the operation. The long list of enthusiastic users of this procedure and the fact that intra-ocular inflammation has rarely resulted from its use bespeak its safety in a convincing fashion. While some writers (Gorst, Milner, Naftzger) have failed to note weakening of convergence after excessive recession, many more have seen such weakening, and the majority follow Jameson's carefully studied rule of not setting the insertion behind the equator. Opinion is not so uniform with regard to the external rectus muscle, the insertion of which is about only 2 mm. from the equator. Jameson is firm on this point, yet many feel that it is safe to transplant the external rectus muscle back 4 to 5 mm. While the indications for recession have been fully detailed by Jameson and by Berens, there is no uniformity of opinion on this point in the published reports. Some authors use this procedure as the operation of choice, regardless of the type of squint; many combine it regularly with one of the shortening operations, while still others use it only to weaken the action of a hypertensive muscle. Berens and Jameson believe that with greater experience with the recession operation one does fewer shortening operations. Several writers agree with Jameson's rule that 1 mm. of recession will correct 5 degrees of deviation. The amount of correction from one recession varies greatly (from 10 to 50 degrees). Jameson expects from 20 to 25 degrees of correction from a 5 mm. recession of the internal rectus muscle and from 40 to 50 degrees of correction from a bilateral recession. Recession of the external rectus muscle gives about one third of this effect. Jameson favors one or two recessions for convergent strabismus and reserves the addition of an advancement operation for squint of very high degree or in cases in which the operation must be confined to one eye. Jameson and others have not observed retraction of the caruncle after recession, while others have found this an annoying sequel. It may be important in this connection to note Jameson's technic of not undermining the caruncle. For the details of the technic of the recession operation the reader is referred to Jameson's articles. A number of innovations have been described (Berens, Bickerton, Curdy, F. A. Davis, Gifford, Gorst, Lancaster, Lombardo, Milner, Nugent, Peter, Prangen, Wilkinson), most of which consist of variations in the number and placement of the sutures without alteration of the fundamental principle or plan.

Tucking.—Burch and Grant are the chief exponents of the operation known as tucking, in the period under consideration. In their excellent

article giving their extensive experience with tucking they described their technic in detail and laid down definite rules for the amount of tuck on each muscle. The technic was also described by Speas, who in addition reported on a new instrument. That tucking is relatively safe no one will dispute. Its accuracy is questioned because of the incalculable amount of stretching. Experience seems to be the only remedy for this, which would make one doubt the validity of any figures. Necrosis of the muscle, slipping of sutures and an unsightly lump produced by the tuck in the muscle are other disadvantages reported. The conclusion of the advocates of tucking is that it is ideal for phorias or very small tropias (those up to 15 degrees). Beyond this it is ineffective and its safety would seem to be overbalanced by its disadvantages.

Advancement.—The question of the fate of the advanced portion of a tendon has not been settled. Some observers (Prangen, Horay) believe that the tendon or muscle becomes adherent to the stump, rendering everything in front of it useless. Jameson pointed out that the muscle fibers may function within an adherent capsule. Peter favors advancement over resection because from 5 to 10 degrees more effect can be obtained by including the conjunctiva, the capsule and the muscle in the shortening process; this operation produces less disturbance of the capsule than resection, and it is better to reattach by tendon than muscle. The objections to advancement other than the uselessness of the advanced portion are: the danger of scleral sutures, the difficulty of placing such sutures securely, the risk of torsion and the unsightly thickening that may persist. All but the last may be eliminated by proper technic. A number of surgeons (B. Moore, Onfray, Saint-Martin, Woodruff, Peter) use the advancement operation as the primary procedure in convergent strabismus, performing it on the external rectus muscle of each eye, if necessary. The great majority, however, use it for strabismus of low degrees to strengthen a hypotensive muscle, or for strabismus of higher degrees, in combination with a recession operation or some form of tenotomy. A few (Bickerton, Jameson) hold it in reserve for later operation if recession proves inadequate. Fifteen degrees is about the average correction from one advancement. Judging from the number of reports on this procedure, advancement is the most popular shortening operation today. The technic first described by Worth is the one most frequently followed. Pollock, Lagrange, Peter, Saint-Martin and others have reported variations. The most striking feature of many of the technics is the complexity of the sutures.

Resection.—The resection operation as described by Reese has served as the model from which most of the variations have branched

in this country. The recent literature on this procedure has conceded it a permanent place in the surgery of ocular muscles and has dealt largely with the details of the technic of the operation. The objections raised to the resection operation are chiefly the danger of sutures slipping and the failure to get sufficient effect. The large number of surgeons who have used this method with eminently satisfactory results indicates the rarity of slipping sutures. Failure to gain sufficient shortening is largely a matter of the technic of not placing the sutures sufficiently far back in the muscle, unless one agrees with Peter's dictum that the resection should not extend beyond the tendon. The question of anesthesia assumes importance here, as it is extremely difficult to obtain by local injection the complete anesthesia required for a large resection. The advocates of resection point out that, in addition to its comparative simplicity and the ease of reanchoring the muscle to its original insertion, it obviates largely the disadvantages of advancement, which have previously been mentioned. The placement of the sutures in the Reese operation causes the muscle to be folded under the stump, and the outer margin of the conjunctiva to be carried between the muscle and the stump. This unsurgical closure has given rise to a number of minor variations, two of which will be described briefly. After making a long curved incision near the outer canthus, Berens places the Prince forceps 1 mm. from the point of resection. He measures this with calipers, or with his new forceps on the shank of which is a millimeter scale. Two double-armed sutures are placed, one near each border, 5 mm. back of the forceps, from within. After the resection the sutures are passed through the stump and conjunctiva and tied. The conjunctiva is closed separately. Lancaster uses a longitudinal incision extending from the limbus to the canthus. The capsule is cut along each margin, and the Prince forceps is applied just in front of the point to be reattached. After the tenotomy two double-armed sutures are put through the stump from beneath and then through the muscle back of the forceps from within. The middle third of the muscle is not included in the sutures, to avoid strangulation. The eyeball is rotated toward the muscle, and the muscle is drawn forward with the Prince forceps by the assistant. Thus the sutures may be tied without tension. The excess part of the tendon is then excised, and the conjunctiva is closed with a running stitch.

Cinch Shortening.—In surveying the recent literature on the O'Connor cinch shortening operation one is impressed by the enthusiasm of almost all the surgeons who have reported on it. No other surgical procedure has called forth such extravagant statements from mature ocular surgeons of high standing, in this country, in recent years. The following statements may be cited as examples: "From the standpoint

of efficiency, safety, certainty of action and surgical principles upon which it is based, no operation yet devised can compare" (McCool); "the most perfect mechanical means of straightening an eye at any age" (H. Barkan). Some surgeons prefer it for the smaller deviations, but others believe that it will give as much shortening as the resection or the advancement operations. Peter found it particularly suited for shortening an internal rectus muscle because of the difficulty of doing other shortening operations in the limited space. The objections raised to the cinch shortening operation are the limited effect from the shortening and the marked reaction from the operation. For the exact technic and the arguments in favor of this operation the reader is referred to excellent descriptions, with illustrations, by Barkan, McCool, Hosford and Hicks, and O'Connor. Jackson found it to be the general verdict of all who have given the cinch shortening operation an intelligent and extended trial that it is superior to other shortening operations.

One of the newer and more original contributions to the surgical treatment of strabismus is the method for shortening a muscle by means of the myocampter, perfected by Barraquer. In recent years it has enjoyed considerable popularity in Spain and France. This is a completely bloodless procedure. The conjunctiva, Tenon's capsule and the muscle are grasped with a forceps 10 mm. from the insertion of the muscle. These structures are transferred to the hook of the myocampter and drawn into the instrument by a screw, in a fashion similar to that of most tuckers. The *agrafe,* a silver clip held in the jaws of the instrument, is pinched into position, clamping the entire fold of the conjunctiva, capsule and muscle. This is left in place from three to five weeks. It may fall away in ten days or may remain for six months or more. It is claimed that the *agrafe* is well tolerated. The advocates of this procedure emphasize its extreme simplicity and safety. The operation can always be repeated.

A number of original procedures have been described that may gain acceptance when they have had more thorough trials. Hubbard shortens a muscle by cutting it through well back and overlapping the two portions with mattress sutures. Dudinov's method for shortening a muscle resembles O'Connor's, auricular cartilage being used in place of the dermal sutures. The size of the cartilage controls the amount of correction. To decrease the effectiveness of a muscle, Chevasse has devised two methods. In his "posterior partial myotomy" he places a clamp as far back as possible, and with specially curved scissors he cuts into the muscle from each side, dividing up to four fifths of the fibers. In his "thermoplasty" he blocks off the nerve supply to the anterior segment of the muscle by a transverse band of electrocoagulation in the belly of the muscle. The portion of muscle in front of the band of coagulation becomes a fibrous band. Salvati advocated the

injection of absolute alcohol into the muscle. Dudinov tried this method in two cases and found that, in addition to great pain after the injections, the correction was lost in about three months. Smukler combines with a tuck of the external rectus muscle a stretching of the opposing internal rectus muscle. He accomplishes this by increasing the size of the tuck for three minutes. This apparently weakens or temporarily paralyzes the internal rectus muscle, allowing time for the tuck to adhere. He had uniformly splendid results in a large series of cases.

Operation of Choice.—To decide the operative procedure of choice from reports in the available literature is extremely difficult. One writer of vast experience may declare in favor of the recession operation as the primary procedure, reserving the shortening operation as an auxiliary. Another surgeon of equal standing may take exactly the opposite stand. Heated arguments on this question between ophthalmologists of experience are being carried on in the current periodicals. However, one finds that the majority have taken the middle course and have combined both principles, usually at the same operation and on one eye. The recession operation is the unquestioned favorite among the operations for weakening an ocular muscle; for strengthening a muscle there are several choices, each with some merits, but resection and advancement are most widely employed. It is striking how little real information can be gained from statistical reports of the results of surgical treatment of strabismus. A number of favorable analyses are found in the recent literature, but they are of scant assistance in the evaluation of the various procedures.

Age for Operation.—With the exception of a representative group of French oculists (Lenoir, Onfray), only a few surgeons (Hill and Courtney, Penman, Argañaraz, Georgariou) still favor waiting until near puberty before operating for strabismus. The great majority, however, favor operating as soon as more conservative methods of treatment have been given trial and failed, regardless of age. This means that many children are ready for operation at 3 years of age or earlier, and the consensus in this country is that the operation should be done at this time. Notable among the opinions of European authors is Sattler's emphasis on this point. Dunnington urged early operation to avoid secondary muscular changes, postoperative diplopia and the great physical and mental handicap.

Anesthesia.—The question of the type of anesthesia cannot be separated from that of the proper age for operation. Many English surgeons wait deliberately until local anesthesia can be used so that the effect of the surgical procedure can be determined during the operation by the use of the Maddox rod and graded accordingly. In this country it is generally believed that the advantages of early operation

greatly outweigh this, and general anesthesia has been used increasingly, with satisfactory results. In discussing this subject Dunnington stated that general anesthesia is no handicap if a careful preoperative diagnosis is made. If the patient to be operated on is an adult, there is greater variation of opinion. Some surgeons prefer general anesthesia at any age because of the belief that complete anesthesia by local means is rarely achieved, making a generous resection or advancement difficult or impossible. The successful use of tribromethanol in amylene hydrate has removed most of the objections to a general anesthetic, but this should be combined with a local anesthetic or with small amounts of ether by inhalation. Of the methods for administering a local anesthetic, in addition to the usual instillation of cocaine or one of the newer substitutes, such as butyn, injections of procaine hydrochloride into the muscle or within the muscle cone have met with most favor.

Sutures.—Silk sutures are still being used by the majority of surgeons. However, recent improvements in catgut sutures and particularly in the needles and the method of attaching them to the sutures have caused a number of surgeons to adopt such sutures for routine use. The great advantage of not having to remove the sutures has definitely outweighed the increased cost and the somewhat greater inconvenience in handling them. The sutures of 0000 ten day chromic catgut with atraumatic needles have been established as very satisfactory for work on the ocular muscles.

The great majority of writers on the surgical treatment of squint are on record as favoring conservative treatment before resorting to operation. This consists of full correction of refractive errors as measured after cycloplegia has been induced, correction of amblyopia by various forms of occlusion, and stimulation of fusion. On the last point, however, most of the reports are exceedingly vague. Comparatively few authors have advocated postoperative exercises. The recommendations on this point have been more specific. The stereoscope is the most popular instrument for postoperative treatment, and exercises are usually begun as soon as the bandages are removed. Jameson opposes postoperative exercises as unnecessary and capable of doing harm to a wounded muscle, possibly inducing external deviation through stimulation of the opposing muscle.

PARALYTIC STRABISMUS

Paralysis of the external rectus muscle is treated by means of transplantations of the tendon by a number of authors. Gifford, Beliaev, Goodman, Lancaster and others have described modifications of the Hummelsheim procedure. The principle is that of dividing the superior and the inferior rectus muscle longitudinally and transplanting the

outer portions (one third or one half) to the tendon of the external rectus muscle. The external rectus muscle may be advanced at the same time. Good functional results from this operation have been described. O'Connor utilizes the same principle but claims that he gets greater abduction by transplanting the halves of the vertical tendons farthest from the paralyzed muscle. Wiener confirmed O'Connor's results but described a new technic for obtaining the same effect in a simpler manner and without injuring the vertical rectus muscles. The external rectus muscle is cut off well back (15 mm.), and the anterior portion, still attached to the globe, is split longitudinally into two halves. The upper half is attached to the tendon of the superior rectus muscle and the lower half to the inferior rectus muscle. White pointed out that the transplantation operations are usually accompanied by recession or tenotomy of the internal rectus muscle, which may be a large factor in the result obtained. He is opposed to using the vertical rectus muscles because of the danger of adding a vertical deviation. He favors advancement of the affected muscle with recession of the direct antagonist. If there is a marked secondary deviation he does a recession of the internal rectus muscle of the other eye. For congenital bilateral paralysis of the abducens nerve Chevasse advised bilateral retroplacement aiming at fixed parallelism.

Little has appeared in the literature on the surgical treatment of paralysis of the internal rectus muscle. The most widely accepted treatment for this condition is shortening of the paralyzed muscle. In a case in which there was paralysis of the internal and the inferior rectus muscle and the inferior oblique muscle after a stab wound Peter was able, after recession of the external rectus muscle and tucking of the internal rectus muscle, to obtain good lateral rotation by transplanting the superior oblique muscle. Through an incision in the upper lid he cut the superior oblique muscle from the globe, opened the trochlea, brought the tendon down through a hole in Tenon's capsule and sutured it to the insertion of the internal rectus muscle. The excess part of the tendon was cut off. This procedure is not applicable to an adult with good vision in each eye. Wiener also reported transplanting the superior oblique muscle to the insertion of the internal rectus muscle. In secondary divergence after complete tenotomy of the internal rectus muscle some surgeons have had difficulty in finding the tenotomized muscle. In these cases Bielschowsky, La Rocca and others obtain a cosmetic correction by advancing the capsule, while Jackson has advised transplanting parts of the superior and the inferior rectus muscle.

For paralysis of the superior rectus muscle, shortening the paralyzed muscle or myectomy of the inferior oblique muscle of the other eye was recommended by White and by Dunnington. The choice of operation is determined largely by the amount of overaction of the inferior oblique

muscle and by which eye is used for fixation. The danger of ptosis and of diplopia in the lower field, after shortening of the superior rectus muscle, tips the balance in favor of myectomy of the inferior oblique muscle of the other eye. The myectomy is done near the origin on the periosteum, and approach is either through the skin or through the conjunctiva. Five prism diopters, with the eye in the primary position, is the least correction obtained from tenotomy of the inferior oblique muscle, according to White. For a smaller effect he opens the muscle sheath, resects 1 cm. of muscle and leaves the sheath. Dunnington gave from 10 to 15 prism diopters as the average correction and 22 prism diopters as the upper limit of correction. He has not observed a serious overcorrection. Rugg-Gunn cuts the inferior oblique muscle from its origin and resutures it to the periosteum ¼ inch (0.6 cm.) posterior to its original attachment. O'Connor favors shortening the superior rectus muscle by means of the cinch shortening operation. After advancing the paretic muscle Peter brought the upper halves of the external and the internal rectus muscle up to the stump of the superior rectus muscle. For detailed discussions of this subject the reader is referred to articles by White and Dunnington.

For paralysis of the inferior rectus muscle most authors (Dunnington, White, Berens) have advised resection of the paretic muscle. When there is secondary spasm of the direct antagonist White advised recession of the superior rectus muscle of the same eye. For secondary deviation of the other eye Berens has suggested retroplacement of the superior oblique muscle. Wiener described cutting off the inferior oblique muscle and sewing it to the sclera, nasal to and in front of the inferior rectus muscle.

The operation of choice for a paralysis of the superior oblique muscle is recession of the inferior rectus muscle of the other eye (Berens, Dunnington, Argañaraz, McMullen, White). Dunnington and Berens use tenotomy of the inferior oblique muscle of the same eye when this muscle is overactive. Savin prefers to do a recession of the superior rectus muscle of the same eye. Vasquez Barrière follows the method of Jackson in transplanting the scleral insertion of the superior rectus muscle of the same eye. J. M. Wheeler has given a method for shortening the superior oblique muscle. The superior rectus muscle is temporarily tenotomized, and the globe is rotated downward. A double-armed suture is placed in the middle third of the tendon of the superior oblique muscle, several millimeters from the insertion. The needles are then secured into the superficial sclera, temporal to the insertion, the tendon being thus folded on itself and advanced. Rules for the amount of advancement cannot be given, but it can be graded, and immediate overcorrection is advised. Aurand treated a patient

with complete paralysis of the superior oblique muscle resulting from a stab wound. He divided the external rectus muscle and the inferior rectus muscle longitudinally and advanced the lower and the outer half, respectively, to a point 3 mm. from the limbus at 7:30 o'clock. There was complete disappearance of the squint and the diplopia. Suker has used the same method, with a good functional result.

For paralysis of the inferior oblique muscle, recession of the superior rectus muscle of the other eye has been advocated by a number of authors (Berens, White). Wheeler has described an advancement of the paretic muscle which has given excellent results. Through an incision in the skin the muscle is isolated in the usual fashion and is cut off from the periosteum. It is then carried over the orbital margin and sutured onto the anterior surface of the malar bone with two mattress sutures. A portion of the muscle may be resected at the same time.

The question of the relation between vertical strabismus and lateral strabismus and their surgical treatment has not been settled. It has been fully discussed by White, who, as a rule, favors correcting the vertical deviation first if it is over 5 prism diopters. Others believe that if the lateral deviation is of considerable size there is little hope of a cure from the removal of the vertical deviation alone and that the lateral and the vertical deviation should be corrected at the same time.

SUMMARY

The literature of the last seven years on the surgical treatment of strabismus has been reviewed. An attempt has been made to evaluate the methods in current use and to give the procedure favored by the majority of ocular surgeons. Some of the newer operations are briefly described.

In conclusion, it can be said that there is no unanimity of opinion as to the proper handling of strabismus. Comparably favorable end-results can be achieved by several methods. The procedure of choice for any surgeon is the one with which he is most familiar and in the performance of which he is most skilful.

BIBLIOGRAPHY

Agataton, S. A.: Simplified Method of Muscle Resection for Squint, Am. J. Ophth. 14:1144, 1931.

Alexiadès, S.: Bridled Tenotomy, Arch. d'opht. 50:212, 1933.

Alvaro, M. E.: Results Obtained with Myocampsis in the Treatment of Strabismus. Rev. de ophth. de São Paulo 3:236, 1934; abstr., Am. J. Ophth. 17:1176, 1934.

Amin, M.: Operation of Advancement Without Resection, Bull. Ophth. Soc. Egypt 26:8, 1933.

Argañaraz, R.: Different Clinical Varieties of Strabismus and Their Surgical Treatment, Arch. de oftal. de Buenos Aires 6:127, 1931; abstr., Arch. Ophth. 6:777 (Nov.) 1931.

Asher, K.: Progress in the Treatment of Concomitant Strabismus, Klin. Monatsbl. f. Augenh. 94:704, 1935.

Aurand, L.: New Method of Substitution of Oblique Muscles by Double Oblique Convergent Advancement of Their Divided Synergic Muscles, Right External and Right Inferior or Superior, Ann. d'ocul. 167:213, 1930; Arch. d'opht. 47:241, 1930.

Barkan, H.: O'Connor Operation for Strabismus, Am. J. Ophth. 14:249, 1931.

——Barkan, O.; Randel, H. O., and Smith, H. G.: Squint: Its Physiopathology and Surgical Treatment, Arch. Ophth. 5:691 (May) 1931.

Barker, C. B.: A Plea for Early Treatment of Cross-Eyes, J. Oklahoma M. A. 24:101, 1931.

Barraquer, I.: Subconjunctival Muscle Shortening in the Operative Treatment of Squint, An. Hosp. de Santa Cruz y San Pablo 6:170, 1932; abstr., Zentralbl. f. d. ges. Ophth. 28:363, 1933.

Beliaev, I.: Muscle Transplantation in Strabismus, Sovet. vestnik oftal. 6:129, 1935; abstr., Am. J. Ophth. 18:779, 1935.

Berens, C.: Recession for Strabismus, Am. J. Ophth. 14:148, 1931.

Resection Operation for Strabismus, Arch. Ophth. 8:321 (Sept.) 1932; Tr. Sect. Ophth., A. M. A., 1932, p. 294.

Forceps for Use in Surgical Operations on Ocular Muscles, Arch. Ophth. 14:990 (Dec.) 1935.

Surgical Results in Two Hundred and Twenty-Three Cases of Heterotropia, Surg., Gynec, & Obst. 62:538, 1936.

——Losey, R. R., and Connolly, P. T.: Retroplacement for Strabismus, Am. J. Ophth. 12:720, 1929.

——Payne, B., and Kern, D.: Orthoptic Training and Surgery in Hyperphoria and Hypertropia Combined with Lateral Deviations, Tr. Am. Ophth. Soc. 32:245, 1934.

Bickerton, J. M.: Modern Treatment of Squint, M. J. Australia 2:569, 1933.

Bielschowsky, I.: Myomectomy of the Inferior Oblique, Klin. Monatsbl. f. Augenh. 86:430, 1931.

Brown, A. M.: Auxiliary Suture in Muscle Tucking, Am. J. Ophth. 14:595, 1931.

Bufill, P.: Surgical Treatment of Strabismus, Arch. de oftal. hispano-am. 34:65, 1934.

Burch, F. E., and Grant, H. W.: Some Considerations of Muscle Surgery with Special Reference to the Tucking Method, Am. J. Ophth. 14:489, 1931; Tr. Am. Acad. Ophth. 35:63, 1930.

Burleson, J. H., and Moore, J. B.: Medical and Surgical Treatment of Squint, Texas State J. Med. 28:550, 1932.

Carroll, F. D., and Blake, E. M.: Repair Following Operations on Extra-Ocular Muscles, Arch. Ophth. 8:711 (Nov.) 1932.

Castresana, B.: Surgical Treatment of Convergent Concomitant Strabismus, Rev. cubana de oftal. 2:188. 1930; abstr.. Am. J. Ophth. 13:832, 1930.

Chevasse, B.: Effects of Simple Operations on Cases Thought to Be Undoubted Congenital Bilateral Abducens Paralysis, Tr. Ophth. Soc. U. Kingdom 54:33. 1934.

Thermoplasty of Extra-Ocular Muscles and Posterior Partial Myotomy, ibid. 54:506. 1934.

Clothier, J. V.: Conservative Treatment of Strabismus in Children, Eye, Ear, Nose & Throat Monthly **12**:420, 1933.

Cosgrove, K. W.: Cross-Eye, J. Arkansas M. Soc. **28**:168, 1932.

Crisp, W. H.: Excessive Tenotomy, Am. J. Ophth. **13**:1001, 1930.

Croll, L. J.: Proper Treatment of Concomitant Convergent Strabismus, J. Michigan M. Soc. **34**:543, 1935.

Cruise, R.: Symposium on Squint, Tr. Ophth. Soc. U. Kingdom **54**:49, 1934.

Curdy, R. J.: Recession Operation for Strabismus, Am. J. Ophth. **16**:890, 1933.

Davis, F. A.: Modification of the Jameson Recession Operation for Strabismus, Arch. Ophth. **11**:684 (April) 1934.

Davis, W. T.: Modern Concept and Treatment of Concomitant Strabismus, Kentucky M. J. **30**:419, 1932.
　Esophoria and Exophoria as the Cause of Obstinate Asthenopia, Arch. Ophth. **10**:455 (Sept.) 1933.

Delganov, V. N.: Operations for Strabismus, Arch. oftal. **7**:1, 1930; abstr., Am. J. Ophth. **15**:643, 1930.

Denig, R.: Performance of My Advancement Operation, Klin. Monatsbl. f. Augenh. **96**:177, 1936.

Doherty, W. B.: A Serious Complication Following Operation for Squint, Arch. Ophth. **4**:251 (Oct.) 1930.

Dudinov, O.: On the Question of the Treatment of Strabismus After Salvati, Klin. Monatsbl. f. Augenh. **87**:371, 1931.
　Use of Auricular Cartilage in the Surgery of Strabismus, Sovet. vestnik oftal. **8**:250, 1936; abstr., Am. J. Ophth. **19**:721, 1936.

Dunnington, J. H.: Tenotomy of the Inferior Oblique, Tr. Am. Ophth. Soc. **27**:227, 1929.
　Hyperphoria: Its Etiology, Diagnosis and Treatment, Am. J. Ophth. **14**:1140, 1931.
　Clinical Management of Heterophoria, J. M. Soc. New Jersey **28**:1, 1931.
　The Proper Time for Operation in Strabismus, Arch. Ophth. **10**:438 (Oct.) 1933.

Eckold, K.: Concomitant Strabismus in Children, Kinderärztl. Praxis **5**:214, 1934; abstr., Zentralbl. f. d. ges. Ophth. **31**:685, 1934.

Ellington, A. J.: Some Practical Points on Squint or Strabismus, South. Med. & Surg. **93**:818, 1931.

Fischer, E.: On the Question of the Technique of Tenorraphy, Sovet. vestnik oftal. **7**:122, 1935; abstr., Zentralbl. f. d. ges. Ophth. **35**:198, 1936.

Folk, M. L.: An Analytical Study of Squint Surgery, Illinois M. J. **65**:268, 1934.

Franklin, H. L.: Convergent Squint, Its Cause and Treatment, Southwestern Med. **17**:58, 1933.

Fuchs, B. I., and Kolen, A. A.: Uses of Fascia Lata in Correction of Strabismus, Sovet. vestnik oftal. **2**:236, 1933; abstr., Am. J. Ophth. **17**:179, 1934.

Georgariou, P. M.: Alternating Convergent Strasbismus, Klin. Monatsbl. f. Augenh. **96**:183, 1936.

Gifford, S. R.: Muscle Transplantation for Paralytic Strabismus, Arch. Ophth. **2**:650 (Dec.) 1929.
　Some Notes on the Treatment of Strabismus, Brit. J. Ophth. **19**:148, 1935.

Goldenburg, M.: Control of the Tenotomized Muscle, Am. J. Ophth. **14**:648, 1931.

Goodman, E. L.: Tendon Transplantation for Paralysis of the External Rectus Muscle, M. Ann. District of Columbia **56**:264, 1935; abstr., Am. J. Ophth. **19**:927, 1936.

Gorst, P. E.: Controlled Recession Operation, Lancet **2**:291, 1930.

Hallock, E. K.: Early Treatment of the Cross-Eyed Child, New York State J. Med. **31**:745, 1931.

Handelsman, G.: New Strabismus Forceps, Brit. J. Ophth. **19**:271, 1935.

Hardy, W. F., in discussion on Hosford, G. N., and Hicks, A. M.: O'Connor Cinch Shortening Operation for Heterotropia and Heterophoria, Arch. Ophth. **11**:825 (May) 1934.

Harman, N. B.: On Squint and Its Treatment, Practitioner **132**:539, 1934.

Hepburn, M.: Symposium on the Treatment of Non-Paralytic Squint, Tr. Ophth. Soc. U. Kingdom **52**:336, 1932.

Hessberg, R.: On Squint in Early Childhood, Jahrb. f. Kinderh. **134**:15, 1931; abstr., Zentralbl. f. d. ges. Ophth. **27**:337, 1932.

Hill, E., and Courtney, R. H.: Critical Summary of Surgical Experiences in 1934, Am. J. Ophth. **19**:773, 1936.

Horay, G.: Erfahrungen über 700 Muskelvorlagerungen, Klin. Monatsbl. f. Augenh. **95**:289, 1935.

Horner, W. D., in discussion on Hosford, G. N., and Hicks, A. M.: O'Connor Cinch Shortening Operation for Heterotropia and Heterophoria, Arch. Ophth. **11**:826 (May) 1934.

Horvath, B. V.: A New Type of Muscle Suture, Klin. Monatsbl. f. Augenh. **91**:682, 1933.

Suture for Muscle Resection, ibid. **94**:673, 1935.

Hosford, G. N.: Strabismus: The Present Status of Its Treatment, California & West. Med. **43**:143, 1935.

——and Hicks, A. M.: Two Improved Instruments for Use in the O'Connor Cinch Shortening Operation for Heterophoria and Heterotropia, Am. J. Ophth. **16**:528, 1933.

Causes of Reactions Following the O'Connor Cinch Shortening Operation, ibid. **17**:520, 1934.

O'Connor Cinch Shortening Operation for Heterotropia and Heterophoria: A Critical Survey, Arch. Ophth. **11**:814 (May) 1934.

Hubbard, H. V.: Over-Lapping of Rectus Muscles for Correction of Squint, J. M. Soc. New Jersey **28**:915, 1931.

Hulbert, J., and Lebas, J.: Surgical Treatment of Concomitant Strabismus by the Myocampter, Bull. Soc. belge d'opht., 1935, no. 70, p. 36.

Hutcherson, J. K.: Sixty Degree Convergent Strabismus Corrected with Operation, Kentucky M. J. **33**:436, 1935.

Jackson, E.: Cinch Operation for Strabismus, editorial, Am. J. Ophth. **16**:637, 1933.

Jameson, P. C.: Surgical Entity of Muscle Recession, Arch. Ophth. **6**:329 (Sept.) 1931.

Some Essentials and Securities Which Stabilize Operations on Ocular Muscles, ibid. **8**:654 (Nov.) 1932.

The Ligated Suture, ibid. **12**:377 (Sept.) 1934.

Some Observations as to Indications for Advancement and Kindred Operations, ibid. **13**:3 (Jan.) 1935.

Recession Operation, Am. J. Ophth. **19**:689, 1936.

Jenkins, S.: Strabismus: Its Management and Correction, Texas State J. Med. **26**:976, 1931.

Jervey, J. W.: Strabismus with Some Consideration of Methods for Its Correction, J. South Carolina M. A. **30**:32, 1934.

Kazdan, L.: Problem of the Squinting Child, Canad. M. A. J. **30**:406, 1934.

Lagrange, H.: Principles of the Technique for Muscular Advancement, Ann. d'ocul. **171**:587, 1934.

Lancaster, W. B.: Operation to Shorten the Rectus Muscle with Buried Sutures, Am. J. Ophth. **14**:483, 1931.

　Operations of Ocular Muscles, in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936.

La Rocca, V.: Correction of a Binocular, Post-Operative Divergent Squint, M. Rec. **144**:116, 1936.

Lenoir, M.: At What Age Should Cross-Eyed Children Be Operated on? Prat. méd. franç. **13**:689, 1932.

Lombardo, M.: Single Sclero-Muscular Suture in Jameson's Recession for Squint, Am. J. Ophth. **15**:523, 1932.

López Lacarrère, J.: Modified Myocampter, Arch. de oftal. hispano-am. **33**:277, 1933.

Lordan, J. P.: Orthoptic and Surgical Management of Strabismus, Arch. Ophth. **12**:843 (Dec.) 1934.

　Orthoptic and Surgical Management of Squint, Am. J. Ophth. **18**:961, 1935.

Lyle, D. J.: Strabismus in Children, Arch. Pediat. **48**:619, 1931.

MacCallan, A. F.: Treatment of Squint, Practitioner **130**:48, 1933.

McCool, J. L.: Some Original Experiments with the O'Connor Muscle Shortening Operation, Am. J. Ophth. **13**:491, 1930.

　Ocular Muscle Operations, California & West. Med. **35**:189, 1931.

McMullen, W. H.: Ocular Torticollis, Tr. Ophth. Soc. U. Kingdom **54**:39, 1934.

Markovic, A.: Contribution to the Operative Treatment of Squint, Przegl. med. **6**:125, 1931; abstr., Zentralbl. f. d. ges. Ophth. **25**:679, 1931.

Mayou, Stephen: Discussion of the Surgical Treatment of Unusual Forms of Squint and Heterophoria, Lancet **1**:848 (April 21) 1934.

Melanowski, W. H.: On Squint and Its Treatment After the Practice at the Warsaw Eye Clinic, Klin. Oczna **9**:156, 1931; abstr., Zentralbl. f. d. ges. Ophth. **27**:338, 1932.

Mérigot de Treigny, M., and Subileau, J. M.: Subconjunctival Muscular Shortening with the Help of the Myocampter in the Operation for Strabismus, Bull. Soc. d'opht. de Paris, February 1933, p. 76.

Merrill, H. G.: Choice of Surgical Procedure for Strabismus, Arch. Ophth. **16**:642 (Oct.) 1936.

Milner, J. G.: Some Remarks on the Recession Operation for Squint, Brit. J. Ophth. **19**:448, 1935.

Moncreiff, W. F.: Strabismus Operations, Am. J. Ophth. **13**:999, 1930.

Moore, B.: Operative Treatment of Squint, M. J. Australia **2**:167, 1932.

Moore, R. F.: Operative Treatment of Squint, Tr. Ophth. Soc. U. Kingdom **54**:3, 1934.

Mott, W. C.: Muscle Recession with Tendon Stump and Tenon's Capsule Fixation, Arch. Ophth. **8**:858 (Dec.) 1932.

Naftzger, J. B.: Strabismus, J. Iowa M. Soc. **25**:80, 1935.

Neame, H.: Discussion on Squint, Tr. Ophth. Soc. U. Kingdom **54**:55, 1934.

Noble, W.: Discussion on Squint, Tr. Ophth. Soc. U. Kingdom **54**:60, 1934.

Nugent, O. B.: Recession Operation with Control Suture, Am. J. Ophth. **18:** 744, 1935.

O'Brien, C. S.: Treatment of Squint in Children, Illinois M. J. **58:**150, 1930.

O'Connor, R.: Cinch Shortening Loop in the Surgery of Extra-Ocular Muscles, West. J. Surg. **39:**670, 1931.

Influence on the Operative Results of Ocular Muscle Action Under Sherrington's Law, Am. J. Ophth. **14:**322, 1931.

Letter to the Editor, Objecting to Peter's Statements About the Cinch Operation, ibid. **16:**1009, 1933.

Superior Rectus Shortening to Correct Certain Horizontal Deviations, ibid. **16:**432, 1933.

Tendon Transplantation in Ocular Muscle Paralysis, ibid. **18:**813, 1935.

The Recession Operation: A criticism, ibid. **18:**1137, 1935.

Surgical Correction of Pure Convergence Insufficiency, Arch. Ophth. **14:**896 (Dec.) 1935.

Onfray, R.: Reflections on Three Hundred Cases of Convergent Strabismus, Bull. Soc. d'opht. de Paris, March 1931, p. 182.

Paderstein: Verstärkte Rucklagerung bei Auswärtsschielen (sogenannte Myotomie), Ztschr. f. Augenh. **71:**107, 1930.

Pagani, M.: Piza da strabismo nell'operrazione diraccorciamento, Atto. d. Cong. Soc. oftal. ital., 1935, p. 699; abstr., Zentralbl. f. d. ges. Ophth. **35:**510, 1936.

Parker, F. C.: Todd Muscle Tuck with Modification, Arch. Ophth. **8:**727 (Nov.) 1932.

Pascal, J. I.: The Doctor's Duty to the Cross-Eyed Child, M. Rec. **139:**646, 1934.

Pavia, J. L.: Correction of Squint with the Myocampter, Rev. oto-neuro-oftal **8:**391, 1933; abstr., Am. J. Ophth. **17:**370, 1934.

Penman, G. G.: Concomitant Squint in Children and Modern Methods of Treatment, Proc. Roy. Soc. Med. **26:**1009, 1933.

Perwög, M.: Tenotomy of Both Internal Recti in One Sitting in Convergent Strabismus, Ztschr. f. Augenh. **79:**239, 1932.

Peter, L. C.: Tendon Transplantation for Paralysis of the Superior Rectus, Am. J. Ophth. **13:**869, 1930.

Advancement and Other Shortening Operations in Concomitant Squint, Arch. Ophth. **6:**380 (Sept.) 1931.

End Results in Monocular Esotropia, Brit. J. Ophth. **15:**642, 1931.

Monocular Strabismus, Am. J. Ophth. **15:**243, 1932.

The Recession Operation in Squint, Arch. Ophth. **7:**815 (May) 1932; Am. J. Ophth. **15:**962, 1932.

Recession Operation with Specific Reference to the Author's Suture, Tr. Ophth. Soc. U. Kingdom **52:**520, 1932.

Symposium on the Treatment of Non-Paralytic Squint, ibid. **52:**325, 1932.

The O'Connor Cinch Shortening Operation, ibid. **52:**525, 1932.

Recent Advances in Squint, Kentucky M. J. **31:**319, 1933.

Use of Superior Oblique as an Internal Rotator in Third Nerve Paralysis, Am. J. Ophth. **17:**297, 1934.

Surgical Treatment of Squint, Tr. Indiana Acad. Ophth. & Otolaryng. **18:**100, 1934.

Extra-Ocular Muscles, ed. 2, Philadelphia, Lea & Febiger, 1936.

Petragnini, V.: The Myocampter of Lacarrère in the Surgical Treatment of Strabismus, Boll. d'ocul. **13:**808. 1934: abstr., Am. J. Ophth. **18:**581, 1935.

Pischel, D. K.: A Method of Muscle Shortening (O'Connor) Which Has Rarely Been Used in Europe, Ztschr. f. Augenh. **80**:351, 1933.

Pollock, W. B. I.: Technique of Advancement and of Tendon Lengthening in Strabismus Operations, Brit. J. Ophth. **19**:268, 1935.

Post, L. T.: Etiology and Treatment of Strabismus, J. Pediat. **2**:448, 1933.

Poyales, F.: Myocampter, Arch. de oftal. hispano-am. **33**:333, 1933.

Prangen, A. D.: Surgery of the Rectus Muscles of the Eye: Selection of Operative Procedure by Differential Diagnosis, Tr. Am. Ophth. Soc. **32**:273, 1934.

Surgical Treatment of Strabismus in Relation to Orthoptic Training, Surg., Gynec. & Obst. **62**:520, 1936.

Prins, C. W.: Treatment of Squint, Nederl. tijdschr. v. geneesk. **77**:1937, 1933; abstr., Zentralbl. f. d. ges. Ophth. **29**:655, 1933.

Randel, H. O.: Cinch Shortening Method for Extra-Ocular Muscles, J. Oklahoma M. A. **27**:433, 1934.

Robertson, G.: Simple Suture Needle for the O'Connor Cinch Shortening Muscle Operation, Am. J. Ophth. **17**:150, 1934.

Rowland, W.: Operation for Paralytic Squint, Am. J. Ophth. **14**:343, 1931.

Rugg-Gunn, A.: Hyperphoria Associated with Overaction of the Inferior Oblique Muscle Treated by Recession of the Origin, Arch. Ophth. **15**:150 (Jan.) 1936.

Saint-Martin: On Some Operative Techniques (Advancement), Ann. d'ocul. **173**:292, 1936.

Salvati, G.: Treatment of Strabismus by Injections of Alcohol, Ann. d'ocul. **167**:229, 1930.

The Treatment of Strabismus by Local Injections of Alcohol, Gior. di ocul. **11**:4, 1930; abstr., Zentralbl. f. d. ges. Ophth. **23**:579, 1930.

Sattler, C. H.: Progress in the Treatment of Squint, Fortschr. d. Therap. **7**:81, 1931; abstr., Zentralbl. f. d. ges. Ophth. **25**:266, 1931.

When and With What Means Shall the Treatment of Convergent Squint of Children Be Begun? Ztschr. f. ärztl. Fortbild. **31**:529, 1934; abstr., Zentralbl. f. d. ges. Ophth. **32**:534, 1935.

——and Bielschowsky, A.: In an Operation on a Lateral Muscle, Can an Existing Vertical Deviation Be Corrected Through a Higher or Lower Attachment of the Muscle? Klin. Monatsbl. f. Augenh. **90**:235, 1933.

In Cases of Tenotomy with Marked Over-Effect from Wide Retraction of the Muscle, Where Will the Muscle Be Found? ibid. **90**:235, 1933.

Savin, L. H.: Discussion on Squint, Tr. Ophth. Soc. U. Kingdom **54**:67, 1934.

Concomitant Squint and Its Treatment, Lancet **2**:523, 1934.

Scardapane, F.: Surgical Treatment of Concomitant Strabismus, Saggi di oftal. **5**:164, 1930; abstr., Arch. Ophth. **4**:744 (Nov.) 1930.

Schlodtmann: On Myectomy of the Inferior Oblique, Klin. Monatsbl. f. Augenh. **86**:520, 1931.

Schwabe: Strengthening the Effect of Squint Operation Through Suture Fixation of the Globe, Klin. Monatsbl. f. Augenh. **92**:399, 1934.

Smith, E. T.: Inferior Oblique Tenotomy and Its Indications, Brit. M. J. **1**:374, 1934.

Smith, H. C.: Imbalance of Vertically Acting Extra-Ocular Muscles, Arch. Ophth. **4**:533 (Nov.) 1930.

Smukler, M. E.: Stretching the Opponent Muscle as Part of Routine Treatment of Convergent Squint Due to Errors of Refraction, Am. J. Ophth. **14**:808, 1931.

Soloviev, L.: Sutures in Operations for Strabismus, Sovet. vestnik oftal. **5**:214, 1934; abstr., Am. J. Ophth. **18**:388, 1934.

Speas, W. P.: Shortening an Extra-Ocular Muscle by Tucking, Arch. Ophth. **4**:545 (Oct.) 1930.

Stoewer: Principles in Application of Myectomy of Inferior Oblique, Klin. Monatsbl. f. Augenh. **92**:115, 1934.

Subileau, J. M.: Operation for Strabismus by the Procedure of the Myocampter, Ann. d'ocul. **171**:507, 1934.

Sugiyama, S. S.: Experimental Investigation of Muscle-Tucking by Various Cat-Guts in the Operation for Strabismus, Nagasaki Igakkwai Zassi **12**:409 and 426, 1934; abstr., Zentralbl. f. d. ges. Ophth. **32**:346, 1935.

Suker, G. F.: Traumatic Paralysis of the Left Superior Oblique, Am. J. Ophth. **14**:822, 1931.

Swab, G. M.: Early Treatment of Strabismus, Nebraska M. J. **16**:234, 1931.

Szymanski, J.: A Contribution to the Surgery of Strabismus, Klin. Oczna **12**:388, 1935; abstr., Zentralbl. f. d. ges. Ophth. **32**:653, 1935.

Terrien: Limitations of Tenotomy, Arch. d'opht. **50**:219, 1933.

Thaw, R. F.: Treatment of Strabismus in Children, Am. J. Ophth. **14**:543, 1931. Management of Strabismus, Ohio State M. J. **31**:749, 1935.

Thies, O.: Necrosis of Sclera After Advancement of Internal Rectus; Recovery, Klin. Monatsbl. f. Augenh. **88**:532, 1932.

Thomson, W. H. M.: Concomitant Squint, Canad. M. A. J. **30**:524, 1934.

Townes, C. D.: Management of Heterophoria, Kentucky M. J. **30**:37, 1932.

Travers, T. a'B.: Treatment of Concomitant Squint, Australian & New Zealand J. Surg. **3**:258, 1934.
Concomitant Strabismus, London, George Pulman & Sons, 1936.

van der Hoeve, J.: Operative Treatment of Concomitant Strabismus, Nederl. tijdschr. v. geneesk. **78**:504, 1934; abstr., Zentralbl. f. d. ges. Ophth. **31**:306, 1934.

van Heuven, J. A.: Squint in Children: Comprehensive Summary, Maandschr. v. kindergeneesk. **3**:120, 1933; abstr., Zentralbl. f. d. ges. Ophth. **31**:40, 1934.

Vannas, M.: Fixation Suture in Advancement, Klin. Monatsbl. f. Augenh. **84**:691, 1930.

Vasquez Barrière, A.: Surgical Correction of the Motor Disturbances of the Oblique Muscles of the Eye, Arch. d'opht. **48**:779, 1931; Ann. d'ocul. **169**:205, 1932.

Walker, C.: Remarks on Thermoplasty, Tr. Ophth. Soc. U. Kingdom **54**:524, 1934.

Wheeler, J. M.: Advancement of the Superior Oblique and Inferior Oblique Muscles, Am. J. Ophth. **18**:1, 1935.

White, J. W.: Strabismus in Children, South. Med. & Surg. **93**:804, 1931.
Hyperphoria, Arch. Ophth. **7**:739 (May) 1932.
Indications for the Treatment of Combined Lateral and Vertical Strabismus, ibid. **10**:585 (Nov.) 1933.
Paralysis of the Superior Rectus Muscle, Tr. Am. Ophth. Soc. **31**:551, 1933.
When and How Should One Operate for Convergent Strabismus? Arch. Ophth. **12**:699 (Nov.) 1934.
Congenital Paralysis of the Sixth Nerve, Tr. Am. Acad. Ophth. **40**:193, 1935.

Wilkinson, O.: Surgical Treatment of Concomitant Strabismus, Rev. de ophth. de São Paulo **3**:25, 1933; abstr., Am. J. Ophth. **17**:78, 1934.
Surgical Treatment of Concomitant Squint, Arch. Ophth. **11**:423 (March) 1934.

Wolf, S.: Some Remarks on the Etiology and the Treatment of Convergent Squint, Illinois M. J. **58**:433, 1930.

Wolfe, O.: Double Advancement of External Recti and Exercises for Alternating Squint, Eye, Ear, Nose & Throat Monthly **12**:407, 1933.

Woodbridge, C. L.: Cure of Crossed Eyes, Kentucky M. J. **33**:392, 1935.

Woodruff, T. A.: Early Recognition of Convergent Squint and Its Management, Proc. Connecticut M. Soc. **139**:120, 1931.

Wright, R. W.: Necessity for Early Treatment of Strabismus, Canad. M. A. J. **27**:170, 1932.

Young, G.: Recession of Internal Rectus Muscle, Tr. Ophth. Soc. U. Kingdom **51**:514, 1931.

Discussion on Squint, ibid. **54**:64, 1934.

Zabel, W.: Basic Principle of New Treatment of Squint, Med. Welt **7**:1349, 1933; abstr., Zentralbl. f. d. ges. Ophth. **30**:452, 1934.

Zade: Vortishnadel zur Shieloperation, Ber. ü. d. Versamml. d. deutsch. opth. Gesellsch. **48**:331, 1930.

Obituaries

FÉLIX DE LAPERSONNE
1853–1937

Among the ophthalmologists of the past half-century, none was more highly esteemed by his colleagues than the former head of the ophthalmic clinic of the Hôtel-Dieu in Paris.

Félix de Lapersonne was born in 1853 in Toulouse, where he took his medical training. Subsequently he became an intern and later chief of staff under Panas in the ophthalmic department of the Hôtel-Dieu. After serving as clinical professor of ophthalmology at Lyons, he succeeded Panas in his clinic at the Hôtel-Dieu. This hospital, one of the most ancient in Europe, dating back to the seventh century, is situated in the heart of old Paris on the Isle de la Cité and facing the Place du Parvis Notre Dame. It has a clinic that is so enormous and of such a varied character that students and clinicians are drawn to it from the most remote French possessions.

I remember with keen pleasure the courtesy and consideration with which de Lapersonne, years ago, demonstrated rare tropical diseases affecting the eye to a small group of foreign visitors. His courtesy laid the foundation of a friendship that continued to the end. Some of his students became in time the heads of other important clinics, and such was their devotion to the master that only a word from him was sufficient cause for them to go to infinite trouble to illustrate unusual methods or rare conditions to those who were his friends.

This great teacher was a critical and discriminating observer. There was no branch of ophthalmology which he had not enriched by his contributions. There is no name occurring more frequently in the ophthalmic bibliographies of the past decades than his.

The simple elegance of his manner and his personal charm made him a distinctive figure in any gathering in which he might be present. He visited the United States once only, when he served as vice president of the International Congress of Ophthalmology, held in Washington, D. C., in 1922, and made one of the most important addresses on that occasion. He received a warm welcome and made many friends, but a trans-Atlantic voyage caused him such acute suffering that he could never be prevailed on to attempt it again. He received numerous distinctions, was president of the French Académie de Médecine and a commander of the Legion of Honor.

At the close of the Thirteenth International Congress of Ophthalmology, held in Netherlands in 1929, the International Association for the Prevention of Blindness was organized, of which he was made president. His judgment and organizing skill were of great value in bringing together those whose friendship had been shaken by the great war. An incident which occurred at that time will illustrate his tact and magnanimity. The names were being read of those who would represent the different countries. After the reading was finished, van

FÉLIX DE LAPERSONNE
1853-1937

der Hoeve, who had called the group together, whispered, "Germany seems to be left out. That will not do." I, feeling much the same way, quietly called de Lapersonne's attention to the omission. "Wait," said he, "wait!"

Then, other business having been transacted, de Lapersonne proceeded to say: "The acceptance by a representative from Germay for a position on the council came too late to be included in the list as read. I, therefore, as president of the association, take pleasure in presenting

the name of Professor Axenfeld to represent Germany." The proposal was approved by acclamation, and as de Lapersonne, reaching across the table, grasped the hand of Axenfeld, a thrill went through the group. The animosities which the war had engendered were forgotten. The breach between these two great ophthalmologists was closed.

After his retirement from active work nothing more deeply appealed to de Lapersonne than the widening of the field for the prevention of blindness, a work which his prestige carried throughout the world. When, therefore, the Leslie Dana Gold Medal was presented to him, it gave him great pleasure—not, of course, that any distinction could add to those which his ability and fame had already brought to him, but he was especially gratified to have come to him this evidence of the high regard in which he was held by his friends across the sea. As the medal was pinned on his breast, with the beautiful gesture so spontaneous to the French, he leaned forward and kissed me on each cheek.

In the passing of Landolt, of Abadie, of Morax, and now of de Lapersonne, all who have gone during the past decade, France has lost some of her great teachers, but it is gratifying to know that their pupils, the younger French workers, with newer methods will probably solve many of the problems to which ophthalmologists as yet do not know the answers.

PARK LEWIS.

DR. MED. SØREN HOLTH
1863-1937

Not only ophthalmology in northern Europe but the whole ophthalmologic world has suffered a great loss in the death of Søren Holth on September 23.

Holth was born on July 21, 1863, and died at the age of 74 years, the last of the three great Norwegian ophthalmologists—Johan Hjort, Hjalmar Schiötz and Søren Holth.

SØREN HOLTH, M.D.
1863-1937

Holth was graduated from medical school in December 1891 and received his M.D. degree in December 1896, after having practiced as an ophthalmologist in Drammen for about four years. His thesis, "The Indirect Fixation Blindness of the Eye," was based on the material he collected in Drammen. He settled in Oslo (then called Kristiania),

where he practiced until two years ago, when increasing otosclerosis forced him to retire.

For six years he was first assistant at the university ophthalmologic clinic and acted as locum tenens for Professor Schiötz when the latter was absent. When Schiötz resigned, the chair was offered to Holth, but because of his difficulty in hearing, Holth did not accept the position. Through repeated visits to foreign clinics he continued his studies—even in recent years. He frequently received invitations to demonstrate at various clinics his methods of operating or to read papers, notably at Oxford, Helsingfors, Stockholm and Budapest. The last named city he visited as late as 1932.

His power for work was phenomenal, as witnessed by his one hundred and ten scientific publications. His chief interest was in the study of glaucoma. In his iridencleisis antiglaucomatosa he has left a lasting monument.

Holth was in every respect a courageous man; he never was afraid to try new methods invented and first used by others. He never hesitated to give an opponent the honor he deserved if he found him in the right and himself in the wrong. As a *curiosum* of his immense operative practice it may be mentioned that he, as far as is known, was the only ophthalmologic surgeon to perform a reclinatio cataractae on a lioness—and with good results!

Holth had many interests besides his medical studies. He also had many friends, but he declined to participate in politics. The old poet's words *bene vixit qui bene latuit* are fitting here. A sincere friend has passed, and those who had the privilege of his friendship have suffered a great loss.

HARALD G. A. GJESSING.

Correspondence

TREATMENT OF NEUROPARALYTIC KERATITIS BY CLOSING THE LACRIMAL CANALICULI

To the Editor:—In the September issue of the ARCHIVES, Drs. J. A. MacMillan and W. Cone (Prevention and Treatment of Keratitis Neuroparalytica by Closure of the Lacrimal Canaliculi: Report of a Case, ARCH. OPHTH. 18: 352 [Sept.] 1937) reported a case in which neuroparalytic keratitis following section of the posterior root of the fifth nerve for neuralgia was relieved by cauterization of the lacrimal puncta. Commenting on this result, they stated, "It is comparable to the results described by Beetham after he had closed the ducts in patients with filamentary keratitis." It seems to me that this statement gives too little credit to Beetham, who was the first to employ this procedure to relieve dryness of the eye due to insufficiency of lacrimal secretion. The writers did not state whether or not it was Beetham's communication that led them to employ the procedure in their case.

The writers contended that if after operation for trigeminal neuralgia the tearing is greatly diminished and the conjunctiva congested, the canaliculi should be closed (presumably by cauterization). Since in many cases of this condition the secretion of tears is restored, sometimes at an early date and sometimes only after the lapse of a year, it would seem that such a radical procedure should be postponed as long as possible. Simply tying off the canaliculi would seem to be a better procedure at the outset, and this could be repeated when necessary. When a permanent effect is desired, I have found in cases of filamentary keratitis that extirpation of the canaliculi is the best procedure. After inserting an ordinary pin in the canaliculus, the canaliculus can easily be dissected out, with the sacrifice of little, if any, additional tissue.

The writers stated, "Our studies have convinced us that although the cornea is anesthetic the diminished secretion of tears is the primary factor in the evolution of these corneal lesions." The only evidence, however, that they gave on which they could base this conviction is the satisfactory results obtained in their case and in the case reported by me in which I kept the cornea continually moistened with Ringer's solution. They failed to mention that in the same communication (Verhoeff, F. H.: The Cause of Keratitis After Gasserian Ganglion Operation, *Am. J. Ophth.* 8: 273, 1925) I stated that in six cases of keratitis following operation on the gasserian ganglion I was unable to detect any lacrimal secretion. If they had not ignored this statement, perhaps their conviction would have been still stronger.

The writers described a new method for measuring the rate of secretion of tears, which seems adequate for the purpose desired, but they brought forward no evidence that this method is superior to others. They stated that they have employed this method in the case of "every patient who has been operated on for trigeminal neuralgia in the Montreal Neurological Institute in the past year." Most astonishing is the fact that the only statements they made as to the results they obtained

are that these have varied in the same patient, and that they obtained "definite information as to the presence of secretion and a fairly accurate impression of its amount." They did not state that they employed it in the case which they reported or in any case in which there was keratitis. In other words, except for the evidence furnished by the fact that the keratitis was relieved by cauterization of the puncta in the single case they reported, they offered no new evidence that this type of keratitis is due to dryness.

The writers stated that the findings in their case contradict my theory that the cause of the dryness in cases of the condition in question was due to injury to the large superficial petrosal nerve, since, so they asserted, this nerve was not injured in their case. They gave no evidence, however, that this nerve was not injured, and therefore the evidence that I brought forward as to injury to this nerve in such cases remains as strong as if their case had not been reported. This evidence is so strong that, in my opinion, it proves conclusively that the large superficial petrosal nerve was injured in their case. Presumably their statement to the contrary is based on a supposition of the operator, but such a supposition is without importance in view of the fact that most often, even when the facial nerve is injured during an operation on the gasserian ganglion or one of its roots, the operator is not aware of the fact at the time.

F. H. VERHOEFF, M.D., Boston.

News and Notes

GENERAL NEWS

Annual Course, Research Study Club of Los Angeles.—The Research Study Club of Los Angeles will hold its regular midwinter course in Ophthalmology and Otolaryngology from January 17 to 28, inclusive. The fee, as usual, will be $50. The teaching staff will include Prof. Alfred Bielschowsky, Dr. Edward Jackson, Dr. John O. McReynolds and Mr. Frederick Jobe, on the eye, and Dr. Arthur W. Proetz, Dr. Grant Selfridge, Dr. W. P. Covell, Prof. Vern O. Knudsen, Dr. Simon Jesberg and Dr. Louis K. Guggenheim, on the ear, nose and throat. In addition, there will be twelve instruction courses. The first week will be devoted mostly to the eye, and the second mostly to the ear, nose and throat.

SOCIETY NEWS

Philadelphia County Medical Society, Eye Section.—At the scientific session of the Eye Section of the Philadelphia County Medical Society held on Nov. 4, 1937, at the Wills Hospital, the clinical meeting was in charge of Dr. Alvin W. Howland, chief resident physician. The chief and his assistants presented a contemporaneous group of demonstrations on external disease, pathologic conditions of the fundus, surgical results, research apparatus and clinical facilities, from which members and guests selected their preference.

CORRECTION

In the article by Dr. Avery DeH. Prangen entitled "Some Problems and Procedures in Refraction" (ARCH. OPHTH. 18:432 [Sept.] 1937, an error occurs in the third sentence on page 441. This sentence should read: "One can easily measure the residual accommodative power at the conclusion of a refraction in which cycloplegia was employed by adding a + 3.00 D. lens to the findings and measuring the accommodative range, the difference between the far point and the near point, each expressed in diopters."

The fourth sentence on this page should be omitted.

Abstracts from Current Literature

Biochemistry

DISTRIBUTION OF SODIUM CHLORIDE AND DEXTROSE IN PLASMA AND AQUEOUS. Y. DERRIN, G. JAYLE and P. FRIZT, Compt. rend. Soc. de biol. **125**: 148, 1937.

The authors hold that the theory of dialysis as the source of the formation of aqueous, as suggested by Mestrezat and elaborated by Duke-Elder, requires modification, since the dextrose content of the aqueous is consistently lower than that of the plasma.

J. E. LEBENSOHN.

THE AMYLASE OF THE SUBRETINAL FLUID. H. J. M. WEVE and F. P. FISCHER, Arch. f. Augenh. **110**: 390, 1937.

Having determined that a sugar-forming ferment, which rapidly disappears, often occurs in the subretinal fluid found in cases of retinal detachment, Weve and Fischer have shown that this ferment is a desmoamylase, which appears to originate in the disturbed retina. The quantity of fluid is small and presents obvious difficulties in chemical analysis. The authors describe a new method of micro-analysis for the examination of subretinal fluid. They conclude that the amylase of the subretinal fluid is a peroxydamylase. Its action in the synthesis of sugar is inhibited by cevitamic acid. They believe that measurements of the catalase index of the blood is evidence that the substance is not present in the blood.

E. G. SMITH.

Congenital Anomalies

FAMILIAL MACULAR COLOBOMATA. P. JAMESON EVANS, Brit. J. Ophth. **21**: 503 (Sept.) 1937.

The author found macular colobomas in five members of a family. In this family no defects of the limbs and no other physical defects were found, and apart from the ocular conditions all these persons were normal.

The next generation so far has shown no similar defects. Unfortunately, the previous generation cannot be fully investigated, but the suggestion is that the defect in this family derived from the maternal side. Further observations in the side branches on this side of the family may possibly disclose instances of the same defect.

W. ZENTMAYER.

HEREDITARY DEVELOPMENTAL ERRORS AS CAUSE OF LOWERED RESIS-
TANCE. W. LÖHLEIN, Arch. f. Ophth. 136: 434 (Feb.) 1937.

The assumption that eyes with hereditary developmental errors are
predisposed and less resistant to inflammatory or degenerative diseases
acquired intra vitam is illustrated with three instructive but hardly
conclusive reports of cases.

P. C. KRONFELD.

Cornea and Sclera

A HITHERTO UNDESCRIBED BILATERAL, SYMMETRICAL, SUBEPITHELIAL
CORNEAL OPACITY IN A CASE OF PAGET'S OSTEITIS DEFORMANS:
REPORT OF A CASE. F. PEPPMÜLLER, Klin. Monatsbl. f. Augenh.
98: 1 (Jan.) 1937.

A man aged 62 had suffered from progressive fibrous osteitis
deformans, Paget's osteitis, since 1918. All the bones of the skeleton
became involved, including those of the skull, as is evident from
pictures. Peppmüller observed the eyes since 1929. Two symmetrical
subepithelial opacities in Bowman's membrane were seen in each eye,
one near the temporal, the other near the nasal, limbus. These opacities,
which were oval and brownish were composed of dotted and linear spots.
Fine deposits of melanin were seen in the conjunctiva outside the limbus,
adjoining the areas of the corneal opacities but not connected with
them. They were progressive from the start, while the corneal opacities
remained more or less stationary until 1933. Since that time they had
progressed, finally becoming confluent in the center of the cornea of
each eye. The opacities did not consist of melanin but may represent a
degenerative procedure. It could not be decided whether they were
caused by arteriosclerotic changes or by the specific metabolic changes of
Paget's disease.

K. L. STOLL.

Experimental Pathology

EXPERIMENTAL REPRODUCTION OF OCULAR LEPROSY. A. SANTONASTASO,
Ann. di ottal. e clin. ocul. 65: 321 (May) 1937.

Review of the literature revealed uncertainty as to whether leprosy
can be transmitted to the eyes of animals. Santonastaso inoculated
ten rabbits' eyes with fragments of material from active lesions of the
conjunctiva and cornea of patients with leprosy. Inoculation was made
into the anterior chamber. The eyes were removed at the death of the
animal or after varying periods. Aside from the usual histologic
stains, a careful search for bacteria was made. In most cases a certain
amount of inflammation involving the iris and cornea followed the
inoculation, but the signs of active inflammation usually subsided after
several weeks. In one case the implanted tissue continued to increase
in size, and histologic sections showed that vascular connections had
been established between it and the neighboring iris. In this specimen
alone large numbers of leprosy bacilli were observed. In the other cases
the inflammatory changes observed were considered to be nonspecific
and probably due to absorption of the foreign material implanted in the

chamber. They were accompanied by degenerative changes in the iris and organization of inflammatory products. The stain for bacteria in these cases showed either absence of organisms or a relatively small number of degenerated bacterial forms.

The author concludes that the possibility of inoculating rabbits with leprosy was not definitely determined in his work and that such inoculation if possible occurs only under special conditions. A bibliography is given.

S. R. GIFFORD.

Glaucoma

THE SURGICAL TREATMENT OF CHRONIC GLAUCOMATOUS OCULAR HYPERTENSION. HENRI LAGRANGE, Brit. J. Ophth. 21: 477 (Sept.) 1937.

This article is written in commemoration of the thirty-third anniversary of subconjunctival and limbic sclerotomy. There is a general consideration of the symptoms, pathogenesis and treatment of chronic glaucomatous ocular hypertension. In connection with the pathogenesis Lagrange says:

"The tension retaining the aqueous humor within the eyeball seems to be specially governed by the action of the *pars ciliaris retinae*. In 1883, Boucheron presented to the French Ophthalmological Society an initial contribution to this point, contemporary with the work of Ranvier maintaining that the epithelial elements were superficial glands. These findings are confirmed by pathological histology. The presence in the ciliary body of adenomas, epitheliomas and carcinomas, described by F. Lagrange in his treatise on tumours of the eye, tends to affirm the existence of a differentiated tissue whose structure shows that it is actively concerned in elaborating the aqueous humor.

"The sympathetic is the nerve of secretion, it increases ocular tension when stimulated and the decisive experiment by Pourfour du Petit and Claude Bernard supports the entire physiology of the sympathetic in its relations with the eye. The individual designation of the secretory nerves is due to the work by Dastre and Morat in 1878 and studies showing their autonomy and functional individuality were made by Heidenhain. It evidently remains to define the nature of the profound causes which may precipitate hypersympathicotony, but the foregoing suffices at least to render homage to the theory of 'secretory neurosis' suggested by Donders with reference to glaucoma.

"The alteration in the evacuation of the ocular liquids from the corneoscleral shell becomes added more or less rapidly to the condition of hypersecretion often occurring during the first stages of the disease.

"Secondary trophic disturbances affecting the vessels of the retina and involving the optic nerve add new complications, in varying degrees. These complications result from a vitiated hydrostatic condition and are beyond the control of the mechanism regulating the intra-ocular circulation. All causes which favour vascular sclerosis, especially syphilis and senility, constitute aggravating factors in the progress of these trophic disturbances secondary to hypertension occurring in a closed chamber, which finally produce optic atrophy and the papillary excavation of Schnabel.

"In brief, though from the viewpoint of general pathology there still remain in the pathogeny of glaucoma certain obscure points referring to the true causes of the sympatheticotonic disturbances, the metabolic changes and the liquid exchanges, one fact is shown by clinical observation, namely, that glaucoma is related to nervous, organic, psychic and doubtless humoral instability, and to a so-called diathesis. It has the characters of an 'essential disease,' whose effects vary according to defective functioning which is individual, congenital or acquired with advancing age, the variations constituting factors which have great influence upon the course of the disease. Of its factors, standing in the order of their importance, hypertension occupies the first place and the therapeutic problem is *to prevent vitiation of the liquid exchanges by medical treatment or to avert, by surgical measures, the retention of liquids which is fatal for the neuro-retinal structures.*"

In acute glaucoma the value of iridectomy is unquestionable. Von Graefe has placed in the hands of ophthalmologists a curative procedure which renders his name immortal. The technic of the modified operation of Felix Lagrange as practiced by the author since 1926 is described and illustrated. From results for patients followed for at least one year the conclusion is sincerely warranted that the fistulizing method succeeds in 85 per cent of cases of chronic glaucoma, while iridectomy is successful in an average of from 25 to 30 per cent of cases, according to the best statistics.

W. ZENTMAYER.

CYCLODIALYSIS. L. HEINE, Klin. Monatsbl. f. Augenh. 97: 721 (Dec.) 1936.

Heine refers to two contributions on cyclodialysis which appeared in 1935, both of which demonstrated progress. The first, that of E. von Grósz, showed that cyclodialysis has been more and more practiced at the University Eye Clinic at Budapest during recent years. Cyclodialysis is taking increasingly the place of trephining. The second publication is that by Vannas, whose experiences with this operation were very satisfactory. The opinions and results of other operators, such as Elschnig, Salus and E. Fuchs, are quoted by Heine. He points out that it is possible to operate more exactly with the technic of cyclodialysis than with the trephine operation and emphasizes the superiority of the former over the latter.

K. L. STOLL.

Hygiene, Sociology, Education and History

PROTECTIVE GOGGLES FOR PHYSICIANS. M. H. VIALLEFONT, Arch. Soc. d. sc. méd. et biol. de Montpellier 18: 25, 1937.

Protective glasses for physicians are more needed than is generally realized, in proof of which various cases are cited. In curetting an infected uterus a gynecologist received septic particles in the eye, and severe inflammation resulted. A laryngologist opened a retropharyngeal abscess with a like consequence and loss of considerable visual acuity.

as a final result. A pediatrician opening an ampule of vaccinia virus had some droplets fly in his eye; vaccinia followed, necessitating enucleation of the eye later.

J. E. LEBENSOHN.

MORE UNPUBLISHED LETTERS OF OPHTHALMOLOGISTS ADDRESSED TO HORNER DURING 1856-1885. A. BADER, Klin. Monatsbl. f. Augenh. 97: 787 (Dec.) 1936.

After a short introduction Bader publishes twenty-four letters addressed to Horner from eminent ophthalmologists, such as Troeltsch, A. von Graefe, T. Leber, Michel, Donders, Schweigger, Schmidt-Rimpler, Zehender, Wilhelm Hess, Laqueur, Herman Knapp, Landolt, A. von Hippel and others. Interesting side-lights of a professional and personal nature may be gathered by the reading of these letters. It may be mentioned that Leber described the condition at A. von Graefe's ophthalmic clinic after the death of this eminent man and that H. Knapp referred to extraction of cataract with and without antiseptics in 1881, the year which brought him his hundred thousandth patient.

K. L. STOLL.

Lacrimal Apparatus

DACRYOCYSTORHINOSTOMY. R. E. WRIGHT, Lancet 2: 250 (July 31) 1937.

As the otherwise excellent method of Dupuy-Dutemps proved too lengthy for the busy clinic in India, Wright sought for a quicker and easier method. The new method is characterized by a small facial incision, and the steps are shown in diagrams with explanatory notes. The difficulty in this operation, and particularly in this method, is the suturing of the cut edges in the sac and in the nasal mucosa. Any fine, long needle holder and a large-eyed, fully curved needle measuring from 6 to 7 mm. from the point to the eye will serve. The procedure is made easier by the adoption of lateral hinged flaps. A straight, eyeless needle from 3 to 5 mm. long (with suture material attached) is easiest to pass a point of suture in a small deep hole. The bone in the floor of the lacrimal groove of the sac is removed, and the hinge flaps are approximated and united by two points of suture. The anterior lip of the opening of the sac is then sutured to the periosteum of the crista. The cutaneous wound is sutured.

Wright says that this method is as bloodless as the ordinary operation and is preferable to extirpation in conditions suited for dacryocystorhinostomy, e. g., a dilated sac in a middle-aged patient. A study of the diagrams will explain the method.

ARNOLD KNAPP.

LONGITUDINAL AND TRANSVERSE SUTURE OF THE LACRIMAL CANALICULI: REPORT OF A CASE. K. ASCHER, Klin. Monatsbl. f. Augenh. 97: 770 (Dec.) 1936.

A boy aged 6 years fell against a door-knob and suffered a triangular wound in the upper eyelid. The lid was partly bruised, and the upper canaliculus was severed near the tear point. Ascher closed the wound

with a transverse suture according to the method of Elschnig, after the insertion of a thin probe. The technic is described in detail. The canaliculus was permeable after six days and remained open. Primary suturing of the severed canaliculus is essential, in the author's opinion. Only a transverse suture seems to yield good results, as suggested by Wagenmann in 1913. It appears as if no case in which a successful transverse suture of the *upper* canaliculus was made has hitherto been published.

<div align="right">K. L. STOLL.</div>

Lens

STRIATIONS OF THE AGING NUCLEUS OF THE LENS. H. GOLDMANN, Arch. f. Augenh. 110: 405, 1937.

Goldmann has studied the striations of the aging nucleus of the lens and found that the relations which exist in the anterior part are qualitatively similar to those in the posterior part. Exact measurements were not made. The so-called superficial surface of the aging nucleus is designated by the author as a reflex zone layer of varying thickness, which in many cases may be resolved into its component elementary layers. Goldmann believes that the nucleus increases in size with age and its nuclear or inner border appears to move inward, while the outer border moves toward the capsule. This is apparently the result of repeated superimpositions of recurring bands of disjunction. In the aging lens the reflex zone may be divided into three zones, corresponding to three separate periods of life. These are important in the determination of the time at which the processes occurred in the pathologically changed lens.

<div align="right">E. G. SMITH.</div>

INTRACAPSULAR EXTRACTION OF CATARACT IN DIABETIC PATIENTS: REPORT OF CASES. E. PURTSCHER and H. DIBOLD, Klin. Monatsbl. f. Augenh. 98: 24 (Jan.) 1937.

Extensive studies on intracapsular extraction of cataract in diabetic patients were made jointly by Purtscher, of the ophthalmic division, and Dibold, of the division for metabolic diseases, at the Hospital of the City of Vienna. The authors report in detail on thirty-seven intracapsular extractions of cataract performed on twenty-nine diabetic patients from 1933 to 1936. The data are recorded on tables which refer to the age of the patient, the type of cataract, the condition of the fundus and of the retinal vessels, the complications and results of the operation and the blood pressure and which state whether insulin was given or not. The following complications met with during the operation received special consideration: severe hemorrhages from the conjunctival vessels, dark coloration of the aqueous by pigment of the iris, postoperative rupture of the wound and postoperative irritation of the iris. Simultaneous observation of the metabolic and ophthalmologic conditions yielded the following conclusions:

1. Hemorrhages are apt to occur during the operation, especially during the incision and the formation of flaps, in persons who had to undergo preparatory treatment with insulin, after rapid reduction of

the blood sugar content prior to the operation and in the presence of hemorrhagic retinitis with or without hypertension (increased blood pressure).

2. Accumulation of pigment of the iris in the anterior chamber may be expected in chronic diabetes.

3. Irritation of the iris is imminent in the presence of a high content of blood sugar, in faulty compensation of the diabetes and simultaneous hypertension (high blood pressure).

4. A comparison of diabetic patients with cataract with nondiabetic persons with cataract shows that a much larger percentage of the former have a high degree of hypertension.

5. The proneness of diabetic patients to hemorrhage during the operation is not dependent on the blood pressure.

6. Flooding out of pigment and postoperative irritation of the iris are more frequent in diabetic patients than in patients with normal metabolism.

7. No difference in the result of the operation and in recovery was found between diabetic patients with cataract and healthy patients with cataract after proper preoperative care, and the visual results were practically the same.

K. L. STOLL.

Lids

CHALAZION: ITS TRUE NATURE AND ITS RELATION TO AMETROPIA. G. ROBERT, Ann. d'ocul. 174: 473 (July) 1937.

In a recent article on this subject Solignac mentioned that in 1933 Robert had drawn attention to the influence of ametropia on the development of chalazion. Since the publication of his article the author has had time to verify statements made then and has arrived at the following conclusions:

The chalazion is a retention cyst, appearing always in eyes which show even slight ametropia. Even when large, it will disappear in nearly all cases after a varying period if one corrects the metropia. The chalazion should be cured by optical and medical treatment. It is not an organic tumor. The deformities and modifications of the secreting cells are not surprising when one considers the phenomenon of retention.

S. H. McKEE.

Neurology

SYPHILITIC ARACHNOIDITIS OF THE OPTIC CHIASM. L. HAUSMAN, Arch. Neurol. & Psychiat. 37: 929 (April) 1937.

Hausman has made a careful study of syphilitic chiasmal arachnoiditis per se and in relation to tumors of the brain and other chiasmal lesions. Five cases of syphilitic chiasmal arachnoiditis and seventy-six cases of verified tumor of the brain supplied the data for the analysis. Because of the infrequent reports of this condition in the ophthalmic literature, as contrasted with the frequent reports of nonsyphilitic arachnoiditis, the article should be of interest to every ophthalmologist. As the

author's conclusions so adequately summarize the paper, stressing important points not generally considered, they are quoted directly:

"1. The syndrome of syphilitic chiasmal arachnoiditis may manifest itself clinically in one of two ways: (1) as the classic form of primary atrophy of the optic nerve, with heteronymous visual field defects, which occurred in cases 1, 2, 3 and 4, or (2) as the less frequent form of papilledema without internal hydrocephalus or increased intracranial pressure, which occurred in case 5.

"2. The visual fields in cases of syphilitic arachnoiditis may show hemianopic defects or concentric constriction or both. Blindness may also occur in one or both eyes. The visual impairment resembles that associated with chronic nonsyphilitic cisternal arachnoiditis and expanding lesions of the chiasmal region and when it is associated with serologic evidence of syphilis, even if this is of the blood alone, there is a strong possibility that the underlying condition is syphilitic arachnoiditis of the chiasm. For that reason the interpretation of the hemianopic visual field defects reported in the literature in questionable cases of tabes associated with atrophy of the optic nerve is open to question. The inclination in some quarters to regard the presence of primary atrophy of the optic nerve alone in cases of syphilis, without other neurologic signs, as evidence of tabes is contrary to all sound neurologic teaching. Many of these cases may belong to the group of instances of syphilitic chiasmal arachnoiditis, which would account for the heteronymous peripheral field defects reported in cases of this condition.

"3. Given the classic chiasmal syndrome of primary atrophy of the optic nerve with heteronymous visual field defects, the problem which arises is one not of localization (the chiasmal site of the lesion is evident) but of differential diagnosis, for the etiologic factor may be one of many: (a) an expanding lesion (tumor, cyst or aneurysm), which may be intrasellar, suprasellar or parasellar; (b) arachnoiditis (syphilitic or nonsyphilitic); (c) trauma or (d) heredodegeneration. Syphilis as an etiologic factor should be carefully considered in each case. There were twelve instances of the chiasmal syndrome in the series of cases of combined syphilis and tumor. In three cases positive serologic reactions were shown, and in the remaining seven the condition proved to be a neoplasm in or near the sella.

"The problem of etiologic differentiation may become complicated when certain factors of neoplasm and syphilis exist together in the same case. If a positive Wassermann reaction, either of the blood or of the spinal fluid, is present with the clinical setting of neoplasm of the brain, the likelihood that a tumor other than gumma is associated with syphilis is exceedingly slight. When the factor of syphilis has been eliminated in cases of the chiasmal syndrome of primary atrophy of the optic nerve and visual field defects, the problem of differential diagnosis becomes more difficult, for it is not always possible to differentiate between nonsyphilitic chronic cisternal arachnoiditis and suprasellar tumor with normal roentgenographic findings in the skull. In many such instances of nonsyphilitic primary atrophy of the optic nerve in which exploration has been made for a possible hypophysial neoplasm, the condition has proved to be chronic cisternal arachnoiditis. The relation of hereditary atrophy of the optic nerve (Leber's disease) to chiasmal arachnoiditis is also discussed.

"4. The nature of the pathologic process in syphilitic arachnoiditis is such that it compresses the optic nerves and chiasm. Why this local process around the chiasm should produce atrophy of the optic nerve in one instance and papilledema in another remains obscure. Reference is made to the dilatation of the chiasmal cistern described by others in cases of chronic cisternal arachnoiditis.

"5. The chief problem which confronts both the patient and the physician is the prevention of blindness which may develop within two years. The choice lies between vigorous antisyphilitic medication and surgical intervention to free the adhesions around the chiasm. The former gives little promise of relief; surgical treatment appears to be the method of choice, particularly when blindness is imminent."

<div align="right">R. Irvine.</div>

Primary Demyelinating Processes of the Central Nervous System. A. Ferraro, Arch. Neurol. & Psychiat. 37: 1100 (May) 1937.

This is a special review article summarizing the conditions mentioned in the title. The demyelinization which constitutes the fundamental pathologic changes of diffuse sclerosis has no particular location, extending at times from the frontal to the occipital pole in more or less symmetrical fashion. As a result the entire nervous system is affected, and the patient becomes practically a decerebrate animal.

It is noteworthy that, with other symptoms, in adults initial symptoms generally consist of disorders of vision and acute blindness and occasionally hemianopia, paresthesias, severe ataxia, and sensory disturbances of objective and subjective types. In the fully developed condition acute blindness may appear, associated with optic neuritis and choked disks and partial or total atrophy of the optic nerve. In some cases the fundi are normal. To and fro swinging movements of the eyes have been reported, and external ophthalmoplegia due to involvement of the third nerve is frequent. Involvement of the sixth nerve has been reported. Nystagmus is not common. Paresis of conjugate lateral gaze and hemispasm of the eyelids have been described.

In children disorder of vision as an initial symptom has been noted in 12 per cent of the cases and a combination of disorder of vision and of hearing in 6 per cent.

<div align="right">R. Irvine.</div>

Arachnoiditis of the Optic Chiasm Following Contusion, Without Injury to the Right Palpebral Region. M. Kalt, Puech and Krebs, Bull. Soc. d'opht. de Paris, May 1937, p. 291.

The authors report an interesting case of arachnoiditis of the chiasmal region with unusual findings. After trauma to the right palpebro-orbital region visual difficulties occurred. In addition to central scotomas there existed double hemianopic bitemporal and paracentral scotomas. Intervention seven months after the accident permitted freeing of the optic nerves, which was followed by partial regression of the scotomas and recuperation of visual acuity. Aside from ecchymosis of the skin at the site of trauma, the patient complained only of photopsia at the time of the accident. One month later the visual acuity of

the right eye was 0.2. There was some recovery of vision, but ten weeks after the accident the visual acuity of the right eye was 0.4, while the left eye remained normal. Drawings of the visual fields made during the course of the condition accompany the article, and campimetric fields of the central areas are shown. Operation revealed active arachnoiditis, and vision returned immediately. The authors express the opinion that the lesion was due to belated infection of the meninges following trauma.

<div align="right">L. L. MAYER.</div>

Ocular Muscles

THE PRESCRIBING OF PRISMS IN EVERYDAY PRACTICE. ANDRÉ HUDELO, Ann. d'ocul. **174**: 528 (Aug.) 1937.

Ophthalmologists of different countries have studied from time to time the problem of heterophoria, that is, the subjective disturbance in the convergence-divergence function. In the last few years many authors, English, American and German, and particularly Landolt, in France, have studied the question from both a theoretical and a practical angle.

Hudelo takes up: (1) what class of patients will benefit from the clinical examination for heterophoria; (2) the determination of the state of the convergence-divergence function, with a consideration of the principles, technic and results; (3) the clinical deductions, and (4) the results of examination.

Among seventy-three subjects with functional disorder of vision studied, thirty-two were troubled with only uncertain disturbance of the convergence-divergence function, for which no prisms were prescribed; eight were cured by prisms; eight have not been seen again, and four have had no relief. Seventeen have not appeared to benefit from prisms, for various reasons. The treatment in cases of emmetropia, those of slight ametropia and those of more pronounced ametropia is then discussed, and replies are given to certain possible objections.

<div align="right">S. H. McKEE.</div>

Operations

THROUGH AND THROUGH OPTIC KERATOPLASTY WITH ROTATION OF THE SCAR. R. FRIEDE, Arch. f. Augenh. **110**: 426, 1937.

Friede reports eight cases in which through and through keratoplasty with rotation of the scar was done and evaluates the results in these cases. He discusses the operation and the indications for its use, as well as conditions which must be present in the cornea. The preparation of the patient, the sequela and the complications are delineated. Modifications of the operation are briefly mentioned. The author believes that the operation has much to offer.

<div align="right">E. G. SMITH.</div>

MELIORATION OF LEUKOMA AND REPEATED TRANSPLANTATIONS OF THE CORNEA. V. P. FILATOV, Vestnik oftal. **10**: 637, 1937.

This is a general discussion of melioration of leukoma. Clinical data will be reported later by Filatov's assistants. Filatov believes that

the corneal transplant is a strong physiologic irritant and causes biochemical reconstruction of the opaque cornea by which the corneal elements regain the property of transparency.

Melioration of leukoma, or substitution of part of the leukoma by transparent corneal elements, was first tried by Filatov in 1933. Since the posterior layers of the cornea often remain transparent, he suggests shaving off the anterior layers of the leukoma and substituting for them the transparent anterior corneal layers of the host's cornea. The purpose of the melioration is not to obtain immediate optical results but to create a favorable nutritive field for the corneal transplant which is ingrafted later in penetrating transplantation. If the corneal transplant becomes opaque, another transplant can be substituted, placed near the previous one, so that finally one of the transplants may remain transparent.

Two types of operations for penetrating melioration are described briefly, and three operations for melioration of leukoma by the transplantation of layers are described in detail. Most frequently Filatov uses for this operation the Filatov-Martzinkowsky (FM-2) knife.

O. SITCHEVSKA.

Orbit, Eyeball and Accessory Sinuses

FIVE UNUSUAL CASES OF PROPTOSIS. R. FOSTER MOORE, Brit. J. Ophth. 21: 465 (Sept.) 1937.

The author points out the difficulties in the way of determining the causes of proptosis, especially when the condition is unilateral. Palpation is difficult, and surgical exploration necessitates trauma to muscles and nerves, while removal of the external wall of the orbit is an operation of some magnitude. Roentgenograms are of great value, and their findings may prove conclusive in the way of furnishing evidence of diseases of the sinuses, involvement of bone, pressure due to oxycephaly, or enlargement of the optic canal. They may, however, reveal no abnormality when, as is often the case, soft tissues alone are involved. The pulsation of a prominent eye greatly limits the possible causes. The past history, perhaps even a remote one of removal of a breast or a growth elsewhere, may give the necessary hint that one is dealing with a metastatic deposit in the orbit; the serious illness of the patient, with signs of thrombosis of the cavernous sinus, may make the cause clear, or the site and severity of an injury may make it certain that hemorrhage into the orbit has occurred. There is a group of conditions which are insufficiently recognized because of their rather indeterminate nature.

The suggested causes in the five cases reported are:

In the first, lodgment in the internal carotid artery of an embolus from the auricular appendix or of a plaque from an atheromatous patch in the aorta.

In the second, a misleading red herring drawn across the track of a long-standing arteriovenous aneurysm.

In the third, spontaneous leakage from an atheromatous internal carotid artery

In the fourth, noninfective thrombosis of the cavernous sinus.

In the fifth, necrosis of the internal frontal crest.

The cases are reported in some detail, and comments are made concerning the features leading to the diagnosis.

W. ZENTMAYER.

SYMMETRICAL SYPHILOMA OF THE ORBIT: REPORT OF A CASE. C. PAS-CHEFF, Klin. Monatsbl. f. Augenh. 97: 751 (Dec.) 1936.

The case of a woman aged 43 is reported in whom syphiloma (gumma) developed to such a size in each orbit as to produce exophthalmos, and blindness was present in one eye, resulting from atrophy of the optic nerve. The appearance was that of symmetrical pseudotumors. A piece of the tumor in each orbit was excised; these specimens showed fibrous structure and proliferation of connective tissue, with some hyaline degeneration; the blood vessels were thickened and partly obliterated. Spirochetes were not observed in the tumors.

Antisyphilitic treatment brought prompt recovery, but one optic nerve remained atrophic. In pointing out the symmetrical occurrence of these syphilomas Pascheff referred to symmetrical tuberculomas, which he described some years ago. In contrast to the latter, the syphilomas of this patient had connections with the periosteum of the orbit.

K. L. STOLL.

Physiology

CHANGES OF THE BLOOD PRESSURE IN THE CENTRAL RETINAL ARTERY FOLLOWING CHANGES IN THE POSITION OF HEALTHY PERSONS: REPORT OF CASES. A. KAMOGAWA, Klin. Monatsbl. f. Augenh. 98: 54 (Jan.) 1937.

Kamogawa reports the results of the continuation of his research on the blood pressure in the central retinal artery. The differences in a sitting and in a reclining position were discussed in the previous article (*Klin. Monatsbl. f. Augenh.* 97: 611 [Nov.] 1936).

In the introduction of the present paper the author analyzes the various factors which bring about hypertension. He then gives a résumé of the recent literature and of the clinical research in this field, including a description of the apparatus, the material and the methods of examination. Several tables and twelve charts are added in illustration. After comparing his results with those of other authors, Kamogawa states the following conclusions:

The changes in the blood pressure in the central retinal artery were measured in ten healthy persons for one hour after the transition from a sitting to a horizontal position. Most of the measurements were taken every ten minutes under simultaneous observation of the variations of the general blood pressure, with the exception of one experimental case, in which the measurements were taken every three minutes. Measurements of the intra-ocular pressure were made simultaneously with those of the blood pressure.

He found:

1. The blood pressure in the central retinal artery increased immediately after change from a sitting to a reclining position, but it decreased gradually in systole and diastole during continuous lying down; this decrease lasted thirty minutes.

2. The first curve of the decrease in the blood pressure began ten minutes after lying down and was steep; the second curve showed a slower declination.

3. The average diastolic blood pressure and the average systolic blood pressure were 7.7 mm. of mercury and 9.7 mm. of mercury, respectively, lower one hour after lying down than immediately after lying down.

4. The systolic and the diastolic blood pressure were 3.5 mm. and 2.8 mm., respectively, lower one hour after lying down than in the original sitting position.

5. The declining curves for the systolic and diastolic pressures were parallel.

6. The higher the increase in the blood pressure was immediately after lying down, the steeper was the declining curve during the first stage.

7. It is Kamogawa's opinion that the decrease in the blood pressure after lying down is due to local regulation, especially in the area of the retinal artery and in the blood vessels subject to the splanchnic nerve; the natural decrease in the action of the heart during reclining is contributary.

8. No distinct change in the pulse pressure was observed in the retinal artery.

9. No change in the general blood pressure or in the frequency of the pulse rate was noted.

K. L. STOLL.

Refraction and Accommodation

REFRACTIVE ERRORS AND COLOR OF THE IRIS. R. BASSIN and B. ŠKERLJ, Klin. Monatsbl. f. Augenh. 98: 314 (March) 1937.

Bassin and Škerlj examined the relation of refractive errors, follicular conjunctivitis and blepharitis to the color of the iris in 2,848 boys and 1,964 girls. The ages ranged between 6 and 22 years. The color was recorded according to the charts of Martin; the refraction was determined with the skiascope, and no mydriatics were used. The results of these examinations were as follows: 1. Myopic and hyperopic errors of a medium or a high degree were more frequent in light-colored eyes (light gray and blue) than in those of a darker hue (brown, hazel and grayish hazel). 2. Refractive errors of a slight degree, i.e., myopia up to 2 D. and hyperopia up to 1 D., predominated in darker eyes. 3. Follicular conjunctivitis and blepharitis occurred twice as often in blue and gray eyes as in light gray eyes and about one third more often in brown eyes than in light gray eyes. 4. The findings were evenly distributed in both sexes. 5. Lack of pigment may be a sign of general biologic inferiority, and racial differences may influence the results obtained.

K. L. STOLL.

Retina and Optic Nerve

THE PROGNOSIS IN PAPILLOEDEMA. G. HOLMES, Brit. J. Ophth. 21: 337 (July) 1937.

In this article only that form of papilledema associated with increased intracranial pressure is considered. It is well known that even intense

papilledema may not cause any disturbance of vision, though it is not so generally recognized that vision may remain unimpaired for long periods, in other words, that the papilledema may not be progressive. In cases of this condition there are probably quiescent or slowly growing tumors, permitting adaptation, so that there is no great or sudden rise of pressure. In rarer instances the papilledema subsides without surgical intervention or other specific treatment, and vision remains unaffected. This shows that papilledema as such does not always demand surgical intervention. The more rapidly the congestion and swelling of the optic disk develops, the more intense does the papilledema become and the greater the danger to the sight if the intracranial pressure is not relieved. Swelling of 5 D. or more is usually a danger signal warning against undue delay. Great engorgement of the retinal veins and early and extensive hemorrhage on the surface of the swollen disk may be regarded as the most important measure of the severity of the process.

Contraction of the arteries may be regarded as the first evidence of constriction in the structures in the nerve head by the organizing fibrous and glial tissue, and irreparable damage of the nerve fibers soon follows. Holmes agrees with Paton that transient loss of vision is usually due to compression of the chiasm and optic tracts by the bulging or distended floor of the third ventricle. When this temporary amaurosis occurs in association with a highly swollen disk it is to be regarded as a threat of permanent failure of vision.

When visual acuity is seriously reduced before operation appreciable recovery rarely takes place. Vision may also fail permanently after a decompression operation in cases in which neither the changes in the fundus nor the visual fields suggested the risk of such a catastrophe.

The article should be read in the original.

W. Zentmayer.

Atrophic Recession of the Lamina Cribrosa. A. C. Reid, Brit. J. Ophth. 21: 361 (July) 1937.

A woman aged 70 years had obvious deep cupping and wide exposure of the lamina cribrosa associated with a decidedly subnormal intraocular tension. Vision was 6/6, and the fields were practically full.

There was no history of attacks suggesting raised tension.

Reid contends that this case represents a group in which needless and useless operations are today being done.

The essential thing to bear in mind is that such recession of the lamina cribrosa and even atrophy may occur independently of any rise in the intra-ocular pressure. Three conditions may be envisaged which may combine or, for a time, at least, remain separate, and all three may occur independently of increased intra-ocular tension: (a) primary atrophy of the nerve elements, (b) cavernous atrophy involving both the nerve and the supporting tissue and (c) atrophic recession of the lamina cribrosa. No conception of retraction is needed to explain (c). In this case the picture was a combined one of (b) and (c), i .e., atrophic recession of the lamina cribrosa associated with involvement of the nerve elements. The term "atrophic recession" seems adequate to explain the condition. Such recession is likely to be accompanied by atrophy, first

of the glial elements in front of the lamina cribrosa, thus leading to a wider exposure. To quote Elliot, the problem "is not the simple one of the physical effects of pressure alone." Hence may not the factor other than pressure alone be the blood supply of the glia (central artery) and of the lamina cribrosa (the circle of Haller)?

W. ZENTMAYER.

A CASE OF OGUCHI'S DISEASE IN SWEDEN. K. O. GRANSTRÖM, Klin. Monatsbl. f. Augenh. 98: 77 (Jan.) 1937.

Only a few cases of Oguchi's disease have occurred outside of Japan. Granström records these and adds one case of his own observation. A man aged 25 showed yellowish white spots in the fundus, which appeared perfectly normal after one half hour of adaptation in the dark room. He complained of hemeralopia since childhood, and his sensitiveness to light was considerably reduced when tested with Gullstrand's photoptometer. The visual fields were normal for white, red and blue, and the perception of color was normal. The disks showed no abnormality. In the fundus of each eye a diffused slate-colored, or grayish-whitish yellow, discoloration was noted, which was most distinct in the periphery. The central retinal vessels, which were normal as a whole, seemed to be raised above the level of the retina in some places. Numerous reflexes were seen all over the fundus, including the macular region; the fovea appeared brighter red than usual, resembling its appearance in cases of embolism of the central retinal artery. The design of the choroidal vessels could be observed only after adaptation in the dark room, when the retinal phenomena had practically disappeared.

There was distant consanguinity of the parents. No other member of the family had hemeralopia; the patient's only sister was myopic, but her sensitivity to light was hardly more reduced than in many myopic persons.

The Wassermann reaction was negative, and the patient belonged to blood group O, blood type N. He was of the Nordic type, a mixture of the Svea and the Göta type, with possibly a slight Semitic admixture. Granström is convinced that his diagnosis of Oguchi's disease is correct.

K. L. STOLL.

LIPOID IN THE PIGMENT EPITHELIUM IN RETINITIS PIGMENTOSA: REPORT OF A CASE. R. ASAYAMA and A. TAKAGI, Klin. Monatsbl. f. Augenh. 98: 162 (Feb.) 1937.

The right eye of a man aged 56 became blind as a result of retinitis pigmentosa and simple glaucoma. Atrophy of the optic nerve and edema of the choroid were seen histologically The retina, choroid and pigment epithelium showed the typical degeneration. In the degenerated portion and also in the pigment cells which had migrated into the retina granules of lipoid of varying size were observed. They differed distinctly from granules of fuscin and consisted of free oleic acid. This process inhibited the formation of visual purple, suggesting a relation of this disease

to hemeralopia. The authors concluded that diffused steatosis of the pigment epithelium is not a requisite for the occurrence of hemeralopia. Calcified drusen were present in the pigment epithelium, and some were deposited on the glassy membrane.

K. L. STOLL.

Trachoma

NEW EXPERIMENTAL RESEARCHES IN TRACHOMA. CUÉNOD and R. NATAF, Ann. d'ocul. 174: 433 (July) 1937.

Cuénod and Nataf introduce the subject with a review of the work which they, in association with Charles Nicolle, have been carrying on It is over thirty years since one of them (Cuénod) with Nicolle began the experimental study of trachoma and arrived at certain conclusions, which have been confirmed by recent pathologic examinations. In 1909 and 1911 these authors, continuing their researches, reported on a series of experiments done on the chimpanzee and man and demonstrated that the chimpanzee offers perfect receptivity and that in this animal the lesions are of the same type and run the same course as in man. In the beginning, trachoma is accompanied by neither inflammatory phenomena nor hypersecretion. The best method to bring on infection in the monkey is by means of scarification, and without doubt trachoma may begin in man after the most superficial abrasion of the conjunctiva. Finally, trachoma in the chimpanzee can be communicated by inoculation during a long period of its course; it is contagious in man under the same conditions, and not only at the beginning of the disease, as was formerly believed.

In reporting this new work Nicolle and Cuénod definitely demonstrated the insidious character of the onset of trachoma and the lengthy period that it was contagious. To crown these experiments they demonstrated in 1912 that the agent of trachoma is a filtrable virus.

Cuénod and Nataf describe in detail their experimental work in their study of the virus and the details of the study of the trachomatous tissue and scrapings from the follicles of trachoma, in which they found what they call *plastilles* (drops of plasma) and which they believe to be analogous to the inclusions observed in the epithelial cells by Prowazek and Halberstädter.

At different times they inoculated lice, guarded against any outside contamination, with trachoma virus, and the result was always a veritable swarm of rickettsial elements in the intestines, while the control lice remained normal. On the other hand, in many cases of infantile folliculosis or of nontrachomatous conjunctivitis the inoculation of lice with conjunctival discharge always gave negative results. On the basis of the results of their inoculation experiments with monkeys the authors believe, first, that the trachoma virus may well be classed in the rickettsia family and that it may be more or less transmitted by agents of this family, and, second, that if lice are not the only transmitters of trachoma they may constitute more or less a reservoir for the trachoma virus.

The article is an extensive one and should be read in the original, as it is too long for a complete abstract.

S. H. McKEE.

ETIOLOGY OF TRACHOMA. A. CUÉNOD and R. NATAF, Rev. internat. du trachome 14: 117 (April) 1937.

The authors are convinced that the etiologic agent of trachoma is a rickettsia-like body. They propose the hypothesis that in families infested with lice the children infect their fingers by scratching, and then by rubbing their eyes transfer the infection.

J. E. LEBENSOHN.

Tumors

TUMOR OF THE CHOROID: METASTASIS IN THE LIVER EIGHT YEARS AFTER ENUCLEATION. P. DUPUY-DUTEMPS, Bull. Soc. d'opht. de Paris, May 1937, p. 284.

The patient was first seen in 1928 at the age of 33 years. Visual acuity of the left eye had gradually diminished to perception of fingers at 20 cm. The ophthalmoscope revealed a pigmented tumor of the choroid near the posterior pole with extensive detachment of the retina. The ocular tension was not raised. Enucleation was carried out, and histologic examination confirmed the diagnosis of nevocarcinoma. The patient has been watched carefully since that time. Eight years afterward, in 1936, hepatomegalia was discovered. Attending symptoms were fever, digestive disturbances and pain over the hepatic zone. Exploratory laparotomy was done, and a portion of the hepatic tumor was removed for biopsy. A photomicrograph of a section of the tumor of the eye and one of a section of the tumor of the liver are shown for comparison. Dupuy-Dutemps mentions also ten other cases of tumor of the choroid. In three of these enucleation had been performed sixteen, thirteen and nine years before, respectively. In two other cases no sign of metastasis had appeared after three and four years, respectively. The other five patients showed a metastatic growth in the liver after six months, one year, three years, five years and eight years, respectively. Metastasis in the liver is considered elective.

L. L. MAYER.

TUMOR OF THE OPTIC NERVE. S. BRAUN-VALLON, Bull. Soc. d'opht. de Paris, May 1937, p. 287.

A boy 3 years old was brought to the clinic because of recent deviation of the right eye. The first signs noted by the mother were intermittent deviation downward, together with slight protrusion of the globe. There was no history of trauma or infection. On examination marked deviation downward, with moderate exophthalmos, paralysis of the superior rectus muscle and limitation of motion on abduction and adduction, was noted. The exophthalmos was partially reducible, and there were no signs of inflammation. Ophthalmoscopic examination revealed slight papillary stasis and tortuous and dilated veins but no hemorrhages. The visual acuity of the affected eye was greatly diminished, but owing to the child's age the visual field could not be measured. The left eye was normal. General and neurologic examinations gave no positive findings. Roentgenograms showed failure of the cranial sutures to close and enlargement of the optic canals, especially that on the right.

At operation a tumor encircling the optic nerve, extending from just back of the globe to the optic canal, was found. It was necessary to remove the optic nerve with the tumor. The exophthalmos receded, but the deviation downward and the paralysis of the superior rectus muscle persisted. The histologic diagnosis was epithelioid meningoblastoma invading the nerve fibers. A picture of the child and a photomicrograph of a section of the tumor are included.

L. L. MAYER.

Uvea

DOYNE'S CHOROIDITIS. J. MALBRÁN and E. ADROGUÉ, Arch. de oftal. de Buenos Aires 11: 529 (Sept.) 1936.

A case of Doyne's choroiditis which was not familial and was characterized by honeycombed, white retroretinal deposits in the macular region is reported. A review of similar cases reported by Hutchinson and Tay, Doyne, Mould, Helthouse and Batten, Bikerton, Treacher Collins Lloyd, Stargardt and Behr is given, and distinctive features are pointed out.

C. E. FINLAY.

PURULENT IRIDOCYCLITIS WITH GONOCOCCI WITHIN THE EYEBALL: REPORT OF A CASE. C. VELHAGEN SR., Klin. Monatsbl. f. Augenh. 98: 20 (Jan.) 1937.

A girl aged 20 had slight congestion of the left eye, which changed to iritis, with deposits on Descemet's membrane, after eighteen days. A month later, a hypopyon developed, and a yellow reflex could be observed in the vitreous. In two weeks the lens became totally opaque, the hypopyon increased in size, perception of light failed, and the pain became intolerable, owing to the enormous intra-ocular tension. The latter symptom is unusual in cases of a purulent intra-ocular process. The eye was removed two months after the onset of the inflammation. Gonococci were observed in the lens, ciliary body and ora serrata, although repeated gynecologic examination proved their absence in the vagina, cervix and urethra. The glaucoma was caused by the tumescence of the lens brought on by the bacteria, which invaded the anterior chamber after rupture of the anterior capsule of the lens.

K. L. STOLL.

Therapeutics

LYSOTHERAPY IN DISEASES OF THE EYE. S. M. KHAYUTIN and E. I. ZLATKINA, Sovet. vestnik oftal. 6:713, 1935.

The technic of preparing and of using the lysates is given. A preliminary study of the effect of lysates on the eye was made on a number of rabbits. One hundred patients with various diseases of the eye were treated with lysates. Most of these suffered from chronic diseases in which all other means of treatment were unsuccessful. Lysotherapy gave positive results in ten cases of keratoscleritis. A remarkable improvement was observed in twenty-two patients suffering from phlyc-

tenular keratoconjunctivitis; photophobia, epiphora and the phlyctenae disappeared after from two to four intramuscular injections of lysates. In three of seven patients who suffered from sympathetic ophthalmia, there was a marked improvement in the sympathizing eye. In nine of ten patients with plastic iritis, the pain and irritation of the eye decreased after the third or fourth injection. No favorable results were obtained in vitreous opacities, zonular cataract, chorioretinitis and deep keratitis.

Khayutin and Zlatkina came to these conclusions: 1. Lysotherapy is definitely favorable in scleritis, phlyctenular keratoconjunctivitis and iritis. 2. Lysates in small doses should be included in the treatment of sympathetic ophthalmia as a preventive against threatening blindness. 3. In severe diseases of the eye the dosage of lysates should be small. 4. Injections of lysates are painless and cause no general reaction.

<div style="text-align: right">O. SITCHEVSKA.</div>

Society Transactions

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Clinical Meeting, London, England, Friday, Oct. 8, 1937

MR. W. H. McMULLEN, O.B.E., F.R.C.S., *President*

A CASE OF DEFECTIVE MOVEMENT OF THE LEFT EYE. MAJOR J. BIGGAM, R.A.M.C.

A youth of 18 years who had been training at the Army technical school as a bricklayer was well until one night, when he felt ill, possibly because of something he had eaten. He vomited three or four times, and, in case it should be wanted, a bucket was placed at his bedside. This he struck in the night with his left eye, and a bruise resulted. In the morning when he awoke he felt still unwell and had diplopia. The latter, however, improved slightly for a month or so. All forms of investigation gave a completely negative result, and the Wassermann reaction and the results of neurologic investigation were negative. As there was liability of a fall from a scaffold and injury of himself or other persons, he was "invalided out." In reading a book he tilted his head backward. He is now in full civilian work. He has binocular vision inside a certain area. The opinion of Dr. Gordon Holmes was asked, and Dr. Holmes wrote that the fact that upward and downward movements were involved, rather than movements of individual muscles, suggested that the patient had a supranuclear lesion, and advised carrying out the Roth-Bielschowsky test. This test is considered positive if the eye moves from the position of rest when it is fixed on any object, and the head is moved in the opposite direction. The patient is asked to look at one's face and then slowly flex his head downward. If the lesion is supranuclear, the left eye should move outward.

DISCUSSION

MR. L. H. SAVIN: In the London County Council hospitals last year there were a number of cases of encephalitis which were supposed to be related to the epidemic of influenza.

LENTICONUS POSTICUS. MR. T. HARRISON BUTLER.

Lenticonus posticus has been confused with myopic degeneration of the lens. The latter is due to the fact that the lens is composed of a concentroid mass of nuclei which have a gradually increasing curvature. There may be senile sclerosis of the center of the lens without association with any alteration of the index of refraction, and there is no increase or development of myopia in cases in which that had been nonexistent. Now and then, however, one meets with a case of this

condition associated with a changed refractive index. If some parts of the lens have a higher index of refraction, rays passing through such parts will come to a focus closer to the lens than those which pass through the periphery, resulting in a linear focus. Sometimes one is puzzled by persons declaring that everything they look at appears watery, yet examination by ordinary methods reveals nothing amiss. In such cases, though vision may be 6/4 as examined by test types, some retrobulbar disorder should be suspected. Or the condition in the lens may be one of those which can sometimes be elucidated by the use of the slit lamp. In these cases the myopia may increase with extra-ordinary rapidity.

Lenticonus posticus is an uncommon condition; perhaps one person in ten thousand has it. On looking into the lens with the slit lamp one sees what looks like a globule of oil, and on flashing the light round one makes out a crater-like appearance. One may come across the condition known as lenticonus posticus internus. I show views illus-trating this disorder in two patients, sisters. It was said that there was a family history of the same condition.

A condition which it is necessary to distinguish the foregoing from is glass blowers' or puddlers' cataract. The illustrations I present show peeling off of the zonular lamella of the anterior cataract. The slit lamp shows at once what has happened. The zonular lamella is the outermost part of the capsule, and when the lens is dislocated by violence it is dislocated out of the zonular lamella. The latter remains behind, with the filaments of the suspensory ligament attached to it. When one finds a posterior central opacity combined with peeling off of the zonular lamella, one can almost always say with certainty that it is the result of infra-red irradiation. But I do not think that one can go into a court of law and state that because the patient is a glass blower or a puddler he is therefore suffering from puddlers' cataract; one can say no more than that it is almost certainly so.

I also show pictures of radium cataract. This may result a long time—as much as two years—after the applications of radium. The slit lamp shows large vacuoles just under the capsule. One can see a rarefied space, and definite secondary lenticonus posticus.

(A number of photographs and drawings are demonstrated.)

DISCUSSION

MR. J. FOSTER: I have seen cases of glass blowers' cataract in persons working at the making of glass bottles. One man with this condition had been working on red-hot blooms with a steam-hammer for twenty years. He had only one sound eye, and it showed des-quamation of the capsule, a curling up of the lamellar layer. A peculi-arity of the law is that in cases of this injury compensation can be paid only if the operation had been done within six months after the accident.

DR. A. J. BALLANTYNE, Glasgow: Referring to the changes due to myopia, I had as a patient a woman who had been wearing a —13 D. sphere because of myopia associated with condensation of the nucleus of the lens. She was so discontented with her lens that she had extraction carried out by another physician, but as she then had to wear a +13 D. sphere the contrast was so great that she never became reconciled to it.

Mr. Basil Graves: Optical section of the type of sclerosed nucleus referred to by Mr. Harrison Butler reveals a feature which gives added interest in conjunction with Fincham's recent monograph (The Function of the Lens Capsule in the Accommodation of the Eye, abstr., *Brit. J. Ophth.* **19:** 51, 1935). The article points out certain characteristics relating to the shape, in accommodation, of the anterior capsule, the curvature of which is complicated by shortening of the radius of the central portion. The anterior face shows the same type of curvature, while the posterior face has a relatively uniform curvature. This provides an interesting subject for speculation.

Book Reviews

Medical Treatment of Cataract. By A. Edward Davis, M.D. Price, $3. Pp. 165, with 11 illustrations. Philadelphia: F. A. Davis Company, 1937.

A. Edward Davis, formerly professor of ophthalmology and consultant in ophthalmology of the New York Post-Graduate Medical School and Hospital (Columbia University), has given in a book of one hundred and sixty-one pages an elaboration of the medical treatment of cataract. His object has been to learn the cause of senile cataract and a method of retarding its progress or of clearing up the opacities. His efforts in this field have been persistent over a period of years. The book is divided into twelve chapters. The historical aspects of the subjects are given, followed by a review of the various theories of the etiology of cataract. The importance of early diagnosis is stressed; the pupil should be dilated to afford complete examination of the crystalline lens. If incipient cataract is found, the patient should have the condition explained to him so that he may more readily agree to the necessity of an investigation of his general condition and cooperate in the treatment which is indicated.

The symptoms and signs of cataract are detailed in chapter 4. Under the pathogenesis of cataract, the permeability of the capsule, the effect of toxins, specific and otherwise, calcium and other substances and endocrine deficiencies are considered. A chapter is devoted to spectroscopy of the lens. The greater portion of the book is devoted to the discussion of the treatment of cataract. The effect of correction of errors of refraction is given by J. Burdon Cooper. The dietary treatment is elaborated on, and the use of ethylmorphine hydrochloride, mercury cyanide, iodides, vitamins, glycerin, boric acid and other agents is discussed.

The book, however, seems to be devoted to the defense of the treatment of cataract by the injection of lens antigen as previously advocated by the author in various papers. To establish the rationale of the method Davis quotes: (1) J. Burdon-Cooper ("Subcapsular senile cataract is undoubtedly a chemical decomposition of the lens proteid"), (2) Roemer ("It is a specific metabolic disease of the lens" and "due to a direct toxic action upon the cells and fibers of the lens") and (3) Carrel ("Certain blood cells, e. g., lymphocytes, have a double function, destroying foreign substances and necrotic material and promoting cell proliferation").

Davis himself states: "Lowered nutrition, general and local, together with toxins in the blood, must be held responsible for most senile cataracts." The injection of lens antigen causes the formation of specific antibodies and creates mild leukocytosis which fortifies the healthy fibers of the lens against toxins, while at the same time it destroys or liquefies the dead fibers of the lens and cataractous or necrotic material.

Are these premises acceptable for this thesis? The reviewer grants that specific antibodies may be formed by the injection of lens proteins

and that they are specific for the normal transparent lens proteins, but, not being specific for coagulated or necrotic lens protein, they cannot combine with the latter for its absorption. He thinks that they probably do not reach the lens, protected as it is by the semi-permeable membranes of the ciliary epithelium and of the capsule. The normal aqueous contains little protein and a very low percentage of the antibodies which are present in the blood, but if the antibodies do enter the lens, the reaction would probably be precipitation of some of the remaining transparent lens protein, and this might add to the opacity. The leukocytosis created by injections of lens protein is hardly necessary or desirable in cases of uncomplicated senile cataract. Increased forma-tion of lenticular fibers is not likely to result, as the growth of the lens is at a minimum in the age group of persons with senile cataract. Cataract has never been considered to be the result of, or accompanied by, inflammatory processes of the eye. In cases of uncomplicated senile subcapsular or cortical cataract, floating cells and an increased protein content of the aqueous are not demonstrable. The contention that the blood cells are stimulated to salutary action by injections of lens pro-tein in cases of cataract is not proved.

Davis would apply his treatment only in cases of subcapsular cataract in which vision is better than 20/70. It is not suitable in these cases or in the great number of cases of nuclear or other types of senile cataracts. The book should contain a classification of cataract and state the relative incidence of the varieties. It should define accurately the subcapsular type which is suitable for treatment. The objective changes which occur in the crystalline lens in the development of cataract should be accurately recorded with the aid of the biomicroscope and photog-raphy of the lens, so that these can be used as criteria for the improve-ment which is noted in the cases reported. Well recorded observations of the clearing of vacuoles or globules, water-split sutures or separated lamellae or of the diffuse haze, features which constitute the development of the incipient stage of senile cortical cataract, would be well worth while. Satisfactory objective criteria must be decided and agreed on for the judgment of the clinical effects of the treatment of cataract. A statement of improvement in vision alone is not sufficient.

Davis' statement in the preface "If such knowledge were carried to the general practitioner and if early active treatment were established by the specialist, at least one-half (perhaps, three-fourths) of all operations for senile cataracts as now performed, would be rendered unnecessary," is certainly unfair and misleading to the public. The advertisements by the publisher are particularly misleading and should not be permitted, because the statements contained therein have not been proved. Simply on the basis of the incidence of subcapsular cataract in relation to that of nuclear and other forms of cataract. which are admitted by the author as being not amenable to his treatment, the statement is dis-proved. Dr. Davis' work is an effort to eradicate one of the tragedies of human life and in this respect is to be commended, but his thesis on the arrest and cure of senile subcapsular cataract by injections of lens antigen may not be accepted in book form any more than it has been in his published papers. Perhaps the reason why his work has not been accepted is that he has not set forth the objective data in regard to his cases in a scientific manner. The field of the investigation of cataract, with

reference to its cause, prevention and cure is an important one. Any treatment carried on in a scientific manner is a legitimate experiment. The evaluation of clinical results in this work is difficult, and all available objective criteria should be used. It is to be hoped that in any future reports these demands will be satisfied.

DANIEL B. KIRBY.

Ueber Retinitis unbekannten Ursprungs nebst einer histopathologischen Studie über Misbildungen des Sehorgans. By Carl Heijl, Stockholm. Price, 18 marks. Pp. 40, with 108 illustrations on 50 plates. Jena: Gustav Fischer, 1937.

There is a group of cases of chorioretinitis in which the cause remains undetermined; the condition has been regarded as either inflammatory or degenerative, on the one hand, or due to constitutional causes, on the other.

Formerly most conditions of the eye, including coloboma, microphthalmus and other developmental defects, were regarded as inflammatory. Recently interest in heredity and developmental disturbances has been aroused, and many conditions have been found to be inherited. These changes in the eye follow the same laws as changes elsewhere. Nevus and birthmarks are disseminated deformities of tissue, localized in the mucous membranes, meninges, central nervous system and eyes. Heijl discusses the relation of tuberous sclerosis and neurofibromatosis to the formation of nevus in a most interesting way. The principal topics of this book, however, are retinitis exudativa externa, or Coats' disease; congenital retinal detachment; pseudoglioma (the formation of connective tissue posterior to the lens and persistent hyaloid artery); microphthalmus; orbitopalpebral cysts, and other deformities.

These conditions were always regarded as the result of intra-uterine inflammation, but the author, after his study of ocular rudiments in teratomas, came to the conclusion that there is an unusual analogy between these conditions and the conditions under discussion. He had access to the ophthalmic and pathologic specimens in Stockholm, Upsala and Lund, and the investigation was carried out in Professor Henschen's Pathological Laboratory in Stockholm. The material examined was that in two cases of Coats' retinitis, eight cases of congenital retinal detachment, one case of formation of connective tissue posterior to the lens, one case of microphthalmus associated with coloboma of the iris and two cases of orbital cysts. Heijl also discusses gliosis, rosette formations, pigment cells, anomalous blood vessels and intra-ocular defects of mesodermal origin.

The author concludes that while Coats' disease and congenital retinal detachment were formerly regarded as examples of "ophthalmias," this interpretation is not convincing. A study of problems of heredity and developmental errors has shown that the eye, on account of its complicated structure, is frequently affected by developmental defects and other constitutional infirmities.

In studying this question the author has followed a new scheme, namely, a comparison of the histologic features of pseudoglioma and of severe developmental disturbances which are present in microphthalmus, synophthalmus, orbital cysts and the eyes of teratomas. This has shown

that in all these conditions there are a number of pathologic peculiarities present which can only be defects in development. He concludes that most conditions known as pseudoglioma are developmental defects.

Another question of great importance concerns the nature of the inflammatory and degenerative processes which were present in these cases; the study of this material convinced the author that the degenerative and inflammatory processes which formerly were regarded as the cause are really secondary and result from poor reactive powers of deformed tissue and from its reduced power of resistance. The author also endeavors to explain the occurrence of developmental defects in the glia, the neural epithelium and pigment epithelium.

The book is illustrated with 108 photomicrographs on 50 plates, in which the pathologic changes in these conditions and in other developmental defects are shown.

Heijl has written an excellent and thoughtful treatise on a difficult subject, which suggests a new explanation of important conditions and should stimulate further investigation.

                                                    ARNOLD KNAPP.

Diseases of the Nose and Throat. By Sir St. Clair Thomson, M.D., F.R.C.P. (London), F.R.C.S., LL.D. (Winnipeg), Médecin Diplome en Suisse, Emeritus Professor of King's College Hospital and the Royal Ear Hospital, London, and V. E. Negus, M.S. (London), F.R.C.S., Surgeon for Diseases of the Nose, Throat and Ear, King's College Hospital, London. Fourth edition. Price, $14. Pp. 976, with 402 illustrations. New York: D. Appleton-Century Company, Inc., 1937.

Since the publication of the first edition twenty-five years ago, this work has been regarded as one of the most acceptable on the subject of rhinolaryngology. The fourth edition now appears, thoroughly revised yet maintaining the original plan of Thomson.

This is a book of personal experience, written in a delightful style, offering a valuable and safe guide for the physician engaged in this specialty as well as constituting a satisfactory work of reference. The text is in large and small type in accordance with the importance of the subject. The numerous bibliographic references are to be found as footnotes at the bottom of each page instead of at the end of the chapter—a method which has much in its favor.

The chapters on sinus disease and the larynx are fully presented. The part on peroral endoscopy has been entirely rewritten. Neoplastic growths everywhere in the upper respiratory tract have been accorded a prominent place, with especial reference to removal both by surgical intervention and by diathermy. The subject of syphilis occupies less space than in former editions, as does also that of diphtheria and that of the once important method of laryngeal intubation. More exact means of diagnosis and treatment have brought about these changes. The elusive subject of allergy has not been discussed in much detail, perhaps because the English mind has not yet accepted the protean manifestations of this disease which have been admitted on this side of the Atlantic.

Of especial interest to the ophthalmologist is the part on the treatment of the various foci of infection, those circumscribed regions in which pathologic microorganisms proliferate, giving rise to systemic or local disturbances of serious import.

A septic focus in a tonsil may be suspected when a creamy drop or two of pus can be expressed from the fossa, when the tonsil has a congested sodden appearance and likewise when there is a reddish color of the injected anterior pillar, often accompanied by enlargement of the cervical glands.

Bacteriologic examination cannot be relied on, for one can often procure hemolytic streptococci in pure culture from material from a person in absolute health. The results of tonsillectomy for secondary infection are therefore uncertain.

According to the authors, in less than 4 per cent of the cases of retrobulbar optic neuritis is the condition found to be caused by sinusitis. Many patients recover spontaneously, or the condition may occur as an early symptom of multiple sclerosis. However, if one naris is stuffy and pus can be seen above a red or swollen middle turbinate, it is justifiable to uncap the ethmosphenoid cells.

Attention is drawn to the fact that in the operation for mucocele the bony walls are often eroded, with exposure of the dura. A drainage tube should be introduced down through the nose and retained in situ for a week or more after removal of the cyst.

All malignant growths in the sinuses and orbit should be treated surgically as far as possible, and removal should be followed by irradiation.

To review a book of this character is indeed a pleasure.

HAMPTON P. HOWELL.

The Ocular Fundus in Diagnosis and Treatment. By Donald T. Atkinson, M.D., F.A.C.S. Cloth. Price, $10, net. Pp. 259, with 106 engravings, including 58 colored plates. Philadelphia: Lea & Febiger, 1937.

In this book, which is intended for the use of surgeons, obstetricians, neurologists and other than ophthalmologists, Atkinson has endeavored to "outline the general characteristics of the ocular fundus, in health and disease." As he believes that "realism and accuracy are likely to be enhanced in illustrations done by the ophthalmologist, who himself studies the details of pathology in each case," the book becomes the only one of its kind in English in which the author has himself made the illustrations.

The text consists of eight chapters, dealing in turn with the use of the ophthalmoscope, the normal fundus, pathologic changes in the retinal vessels, the optic nerve, the vitreous, the retina, the choroid and, finally, ophthalmoscopic changes in special diseases. These chapters contain forty-eight schematized drawings in black and white. In connection with most of the various pathologic entities there is a brief section devoted to treatment. These sections serve only to make ambiguous the title of the book. They might well have been omitted.

The colored plates are fifty-eight in number, and it is presumably by these that the usefulness of the book is to be judged. When it is

remembered that the book is not intended primarily for the use of ophthalmologists, it would seem that some objection might be raised in regard to the lesions selected for illustration. Of the fifty-eight plates, eight represent normal fundi. Of the remaining fifty plates, more than thirty illustrate cases interesting enough to have been referred to the author. The chief difficulty which besets the physician who makes only incidental use of the ophthalmoscope is not the recognition of choroidal leprosy, a dipterous larva in the vitreous, Cysticercus cellulosae or even angioid streaks. His great problem is to recognize a normal fundus or to recognize and appraise slight departures from the normal. In this respect Dr. Atkinson's pictures will afford little help, for the conditions portrayed, when not of inherent rarity, become unusual because of extreme severity or extent.

Finally, and perhaps most important of all, Dr. Atkinson has failed to impart a sense of realism, as far as the reviewer is concerned. On the contrary, many of the plates give rise to a decided impression of artificiality. The hemorrhages and exudates depicted seem, almost without exception, much too obvious. On the other hand, nowhere do attenuated arteries seem sufficiently attentuated. The colors of the atrophic nerve heads, especially of those showing glaucomatous atrophy, are unusual, while the picture of the coloboma of the optic nerve comes perilously close to being unrecognizable. The author has undoubtedly expended much time and effort to produce this book. The reviewer believes it fair to say, however, that the field covered has been covered before and covered much better.

O. P. PERKINS.

Directory of Ophthalmologic Societies *\

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

Honorary President: Prof. F. de Lapersonne, 217 Fauborg St. Honoré, Paris.
President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the President, Dr. P. Bailliart.

INTERNATIONAL OPHTHALMOLOGIC CONGRESS

Secretary: Dr. E. Marx, Costzeedijk 316 Rotterdam, Netherlands.
Place: Cairo. Time: Dec. 8-14, 1937.

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. P. E. H. Adams, 6 Holywell, Oxford.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

GERMAN OPHTHALMOLOGICAL SOCIETY

President: Prof. Lohlein, Jena.
Secretary: Prof. A. Wagenmann, Heidelberg.

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. T. E. Ashdown Carr, 34 Charnwood St., Derby, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.
Time: Oct. 1, 1937.

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Prof. Dr. Sayed Abdel Hamid Soliman Pasha, Faculty of Medicine, Cairo.
Secretary: Dr. Abdel Fattah El Tobgy, 3 Midan Soliman Pasha, Cairo.
Time: March 1938.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. Gordon M. Holmes, 9 Wimpole St., London, W. 1.
Secretary: Mr. J. D. M. Cardell, 27 Weymouth St., London, W. 1.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Oxford Ophthalmological Congress

Master: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.

Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.

Time: July 7-9, 1938.

Palestine Ophthalmological Society

President: Dr. Arieh Feigenbaum, Abyssinian Str. 15, Jerusalem.

Secretary: Dr. E. Sinai, Tel-Aviv.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.

Secretary: Dr. J. Sobański, Lindley'a 4, Warszawa.

Place: Lindley'a 4, Warszawa.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Ransom Pickard, 31 East Southernhay, Exeter, England.

Secretary: Dr. A. Rugg-Gunn, 35 Harley St., London, W. 1.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris, 7è.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm, Sweden.

Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö., Sweden.

Tel-Aviv Ophthalmological Society

President: Dr. D. Arieh-Friedman, 96 Allenby Str., Tel-Aviv.

Secretary: Dr. Sadger Max, 9 Bialik Str., Tel-Aviv.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung.

Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. Parker Heath, 1553 Woodward Ave., Detroit.

Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.

Place: San Francisco. Time: June 13-17, 1938.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.

Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Ophthalmological Society

President: Dr. Frederick H. Verhoeff, 243 Charles St., Boston.

Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.

Place: San Francisco. Time: June 9-11, 1938.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.

Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. E. S. Schmidt, 107 E. Walnut St., Green Bay.

Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.

New England Ophthalmological Society

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. William P. Beetham, 5 Bay State Rd., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. William D. Donoher, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: Victoria, B. C. Time: June 21-24, 1938.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. W. Howe, 740 St. Helens Ave., Tacoma, Wash.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. Frank W. Broderick, 501 Central Trust Bldg., Sterling, Ill.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501—7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. Andre Cortopassi, 703 Second National Bank Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. J. H. Judd, 107 S. 17th St., Omaha, Neb.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.
Place: New Orleans. Time: Nov. 30-Dec. 1-3, 1937.

Southwestern Michigan Triological Society

President: Dr. John Hunter McRae, 26 Sheldon Ave., S. E., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. C. Wearne Beals, Weber Bldg., DuBois.
Secretary-Treasurer: Dr. C. Wearne Beals, Weber Bldg., DuBois.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Charles T. Flynn, 41 Trumbull St., New Haven.
Secretary-Treasurer: Dr. Shirley H. Baron, 309 State St., New London.
Place: New London. Time: February 1938.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. John King, Thomasville, Ga.
Secretary-Treasurer: Dr. Mason Baird, 384 Peachtree St., Atlanta, Ga.
Place: Augusta. Time: May 1938.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. E. E. Holland, 51 S. 8th St., Richmond.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: April 6, 1938.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. C. A. McWilliams, G. and S. I. R. R. Bldg., Gulfport, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: New Orleans. Time: Spring, 1938.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. F. N. Smith, Grand Rapids Clinic, Grand Rapids.
Secretary: Dr. Dewey R. Heetderks, 26 Sheldon Ave., S. E., Grand Rapids.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. Walter E. Camp, 1918 Medical Arts Bldg., Minneapolis.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Edward S. Murphy, Northern Pacific Hospital, Missoula.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Oram Kline, 414 Cooper St., Camden.
Secretary: Dr. James S. Shipman, 542 Cooper St., Camden.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Marvin F. Jones, 121 E. 60th St., New York.
Secretary: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Time: May 10, 1938.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville.
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.
Place: Charlotte. Time: October.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. Rosenberger, 221—5th St., Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, 514—6th St., Valley City.
Place: Bismarck. Time: May 1938.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. George Truluck, Orangeburg.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Kate Savage Zerfoss, 165—8th Ave. N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.
Place: Nashville. Time: April 12-13, 1938.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana, Texas.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.
Place: Fort Worth. Time: Dec. 11 and 12, 1937.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. M. H. Hood, 505 Washington St., Portsmouth.
Secretary-Treasurer: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. James A. Fisher, 501 Grand Ave., Asbury Park, N. J.
Secretary: Dr. William F. McKim, 488 Sanford Ave., Newark, N. J.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron.
Secretary-Treasurer: Dr. C. R. Andersen, 106 S. Main St., Akron.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. William C. Warren Jr., 478 Peachtree St., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Henry F. Graff, 513 N. Charles St., Baltimore.
Secretary: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Secretary-Treasurer: Dr. Mortimer A. Lasky, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Thomas D. Allen, 122 S. Michigan Blvd., Chicago.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Webb Chamberlin, 7405 Detroit Ave., Cleveland.
Secretary: Dr. Walter H. Rieger, Hanna Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. C. E. Silbernagle, 247 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. D. M. Johnson, 327 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Lester H. Quinn, 4105 Live Oak, Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Dr. William Fowler, 1424 Maccabee Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. J. D. Carroll, 102—3rd St., Troy, N. Y.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 306 W. Broadway, Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. L. Allen, 1215 Walker Ave., Houston, Texas
Secretary: Dr. George C. Farrish, 1625 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. C. P. Clark, 24 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. O. S. Gilliland, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. F. C. Hertzog, 117 E. 8th St., Long Beach, Calif.
Secretary-Treasurer: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Isaac H. Jones, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. J. S. Bumgardner, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Max Bornstein, Heyburn Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. Thomas A. Judge, 735 N. Water St., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 208 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. A. G. Farmer, 1040 Fidelity Bldg., Dayton, O.
Secretary-Treasurer: Dr. Rome M. Webster, 663 Reibold Bldg., Dayton, O.
Place: Van Cleve Hotel. Time: 6:30 p. m., bimonthly, first Tuesday from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. Stuart Ramsay, 1496 Mountain St., Montreal, Canada.
Secretary: Dr. J. Rosenbaum, 1396 St. Catherine St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Guy Maness, Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Secretary-Treasurer: Dr. H. F. Brewster, 837 Gravier St., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Society for Clinical Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Jesse Stark, 45 Park Ave., New York.
Place: Squibb Hall, 745 5th Ave. Time: 7 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. A. J. Young, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. Delbert K. Judd, 1020 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Ophthalmological Society of the University of Pittsburgh

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

Passaic-Bergen Ophthalmological Club

President: Dr. John S. Van Winkle, 297 Broadway, Paterson, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Glendon E. Curry, Westinghouse Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351—5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. A. F. Bagby, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third
Monday of each month from October to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. Lawrence T. Post, 508 N. Grand Blvd., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and
scientific meeting 6:30 p. m., fourth Friday of each month from October to
April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio,
Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each
month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Joseph W. Crawford, 490 Post St., San Francisco.
Secretary: Dr. Russell Fletcher, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Fourth
Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. A. Wilkinson, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every
month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Raymond A. Lower, 407 Riverside Ave., Spokane, Wash.
Secretary: Dr. Louis A. Parsell, Paulsen Medical and Dental Bldg., Spokane,
Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of
each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gordon D. Hoople, 110 Medical Arts Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 212 Medical Arts Bldg., Syracuse,
N. Y.
Place: University Club. Time: First Tuesday of each month except June, July
and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. Samuel Mortimer Lyon, 122 Bloor St., W., Toronto, Canada.
Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington,
D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 Eye St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday
in November, January, March and May.

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